THE American JOURNAL OF Surgery

JUNE 1957

THURSTON SCOTT WELTON, M.D., EDITOR

Symposium on Malignant Intracranial Neoplasms

Robert F. Heimburger, M.D., *Guest Editor*

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JUNE 1957

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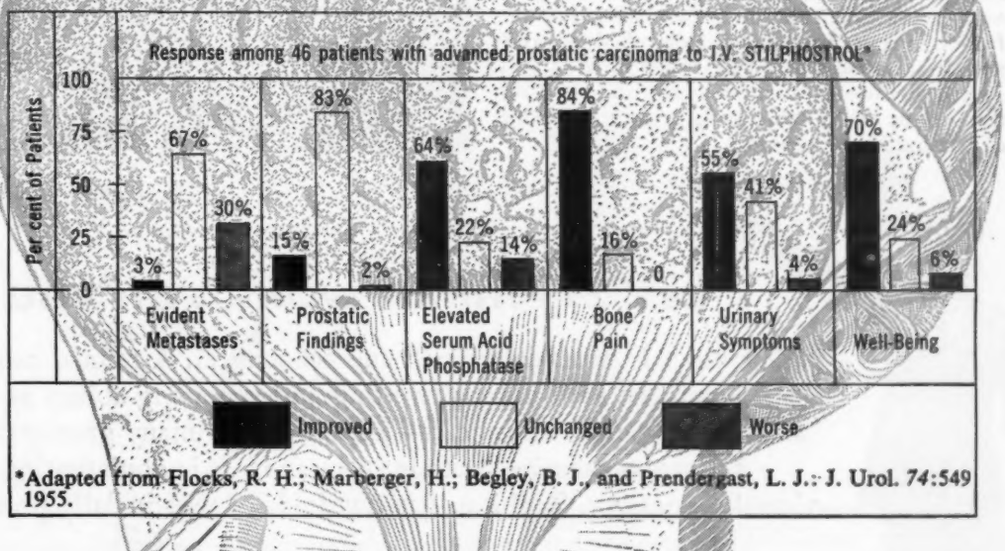

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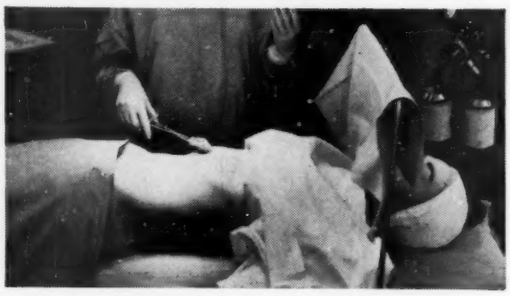

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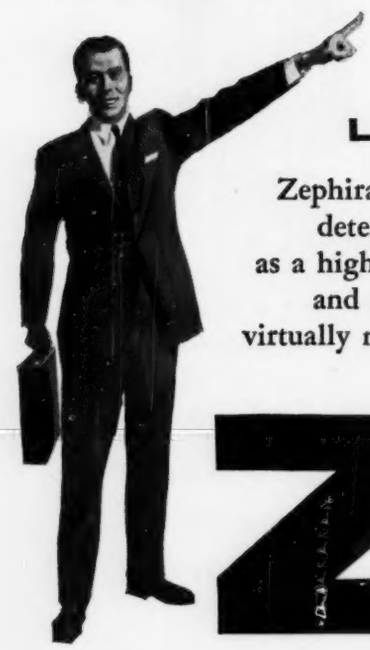

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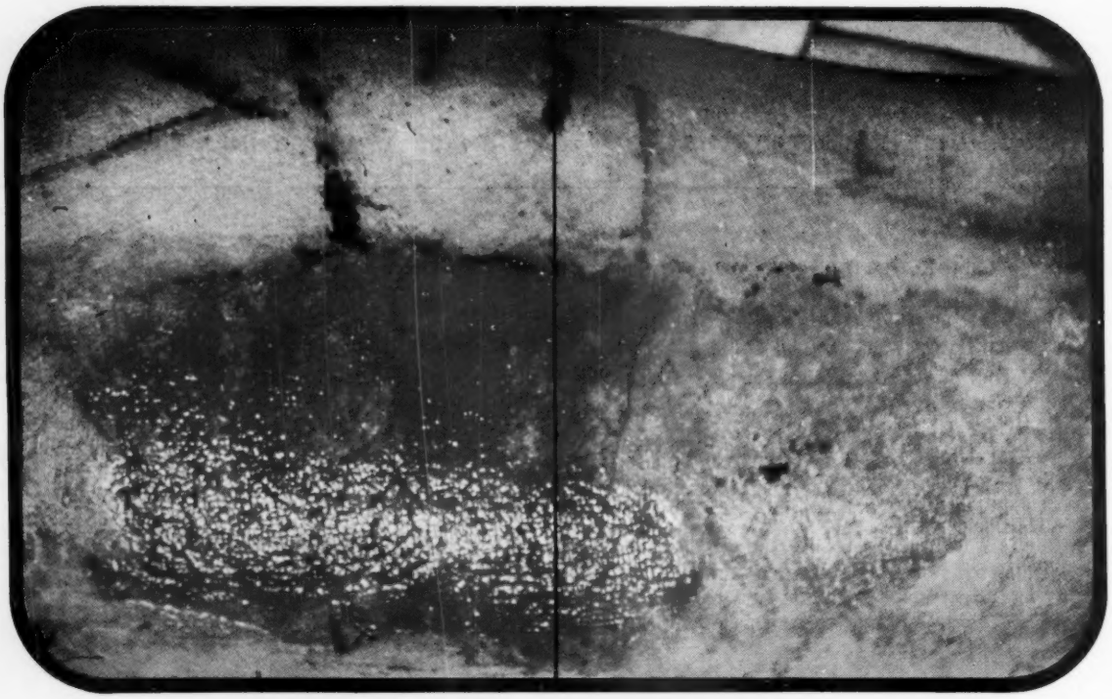

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Congenital Absence of the Appendix 1040

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**Elman, R.: Protein Needs in Surgical Patients, J. Am. Dietetic Assn. 32;524 (June) 1956.*

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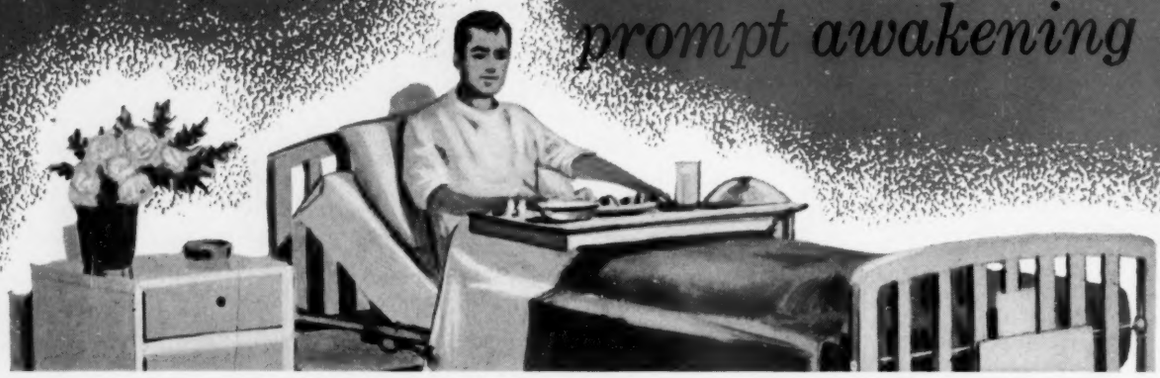

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Bibliography: 1. Hand, J. R.: J. Urol. 75:973 (June) 1956. • 2. Editorial: J. A. M. A. 163: 650 (Feb. 23) 1957. • 3. Lapin, J. H., Klein, W., and Goldman, A.: J. Pediat. 22:175 (Feb.) 1943.

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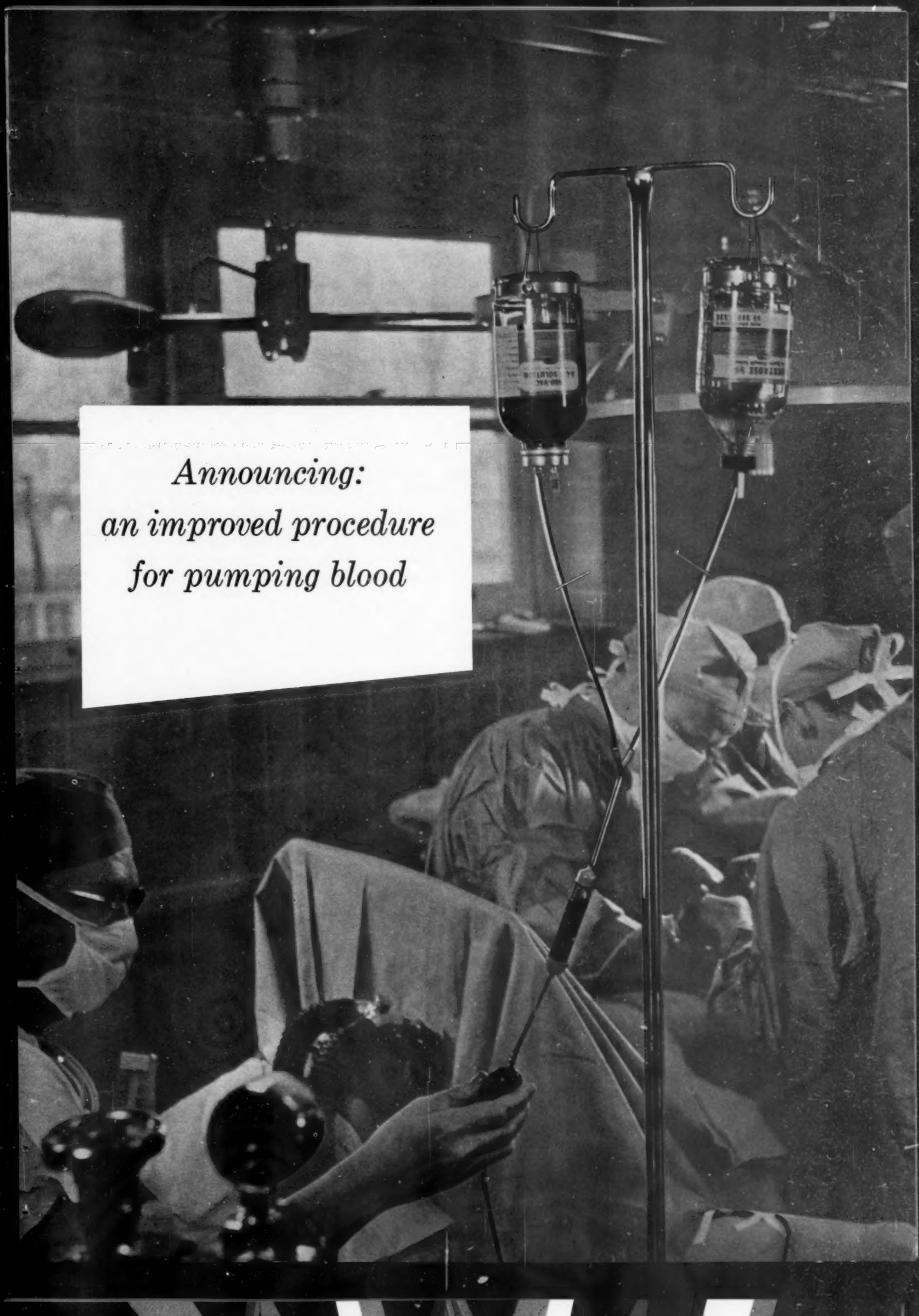
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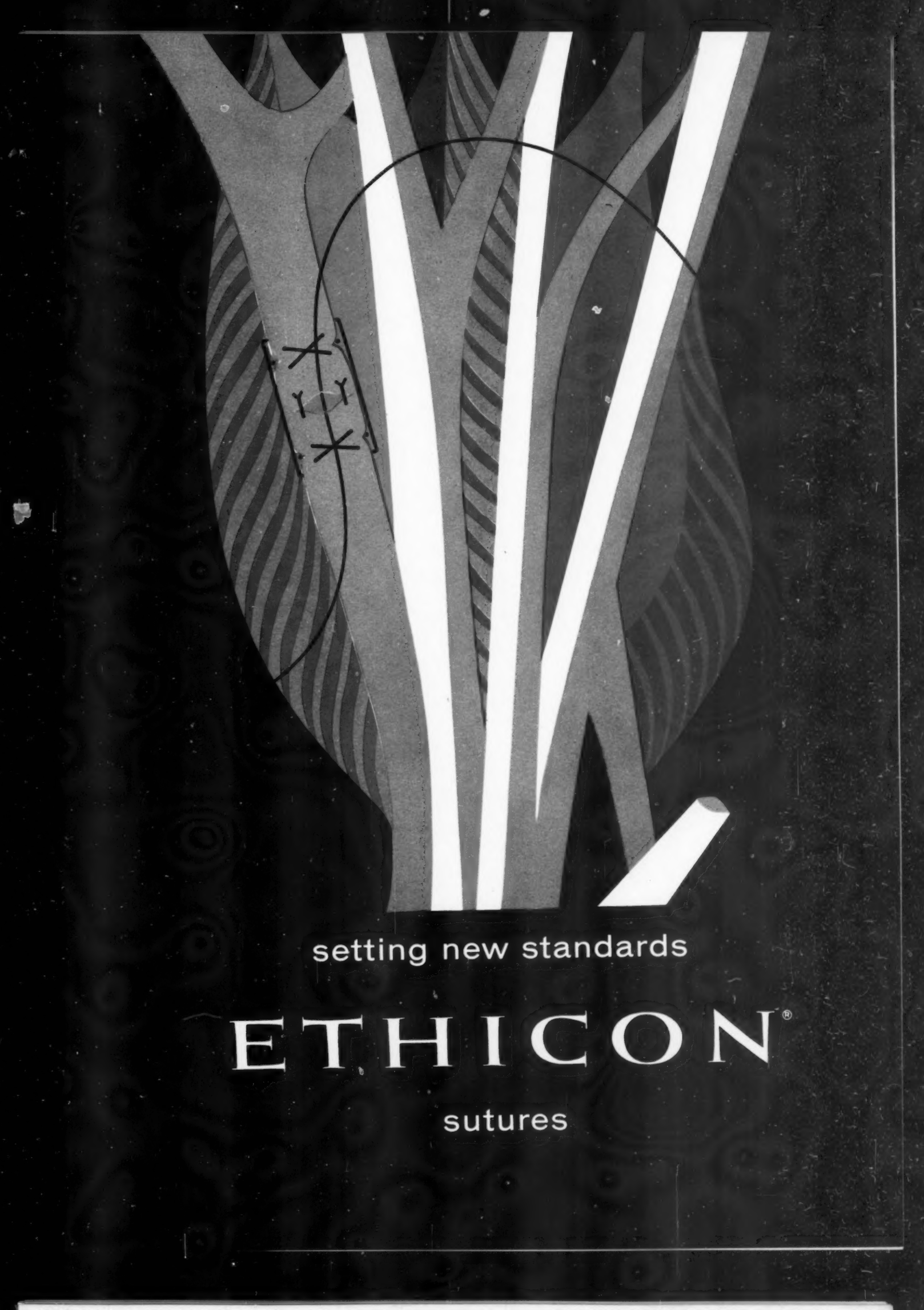
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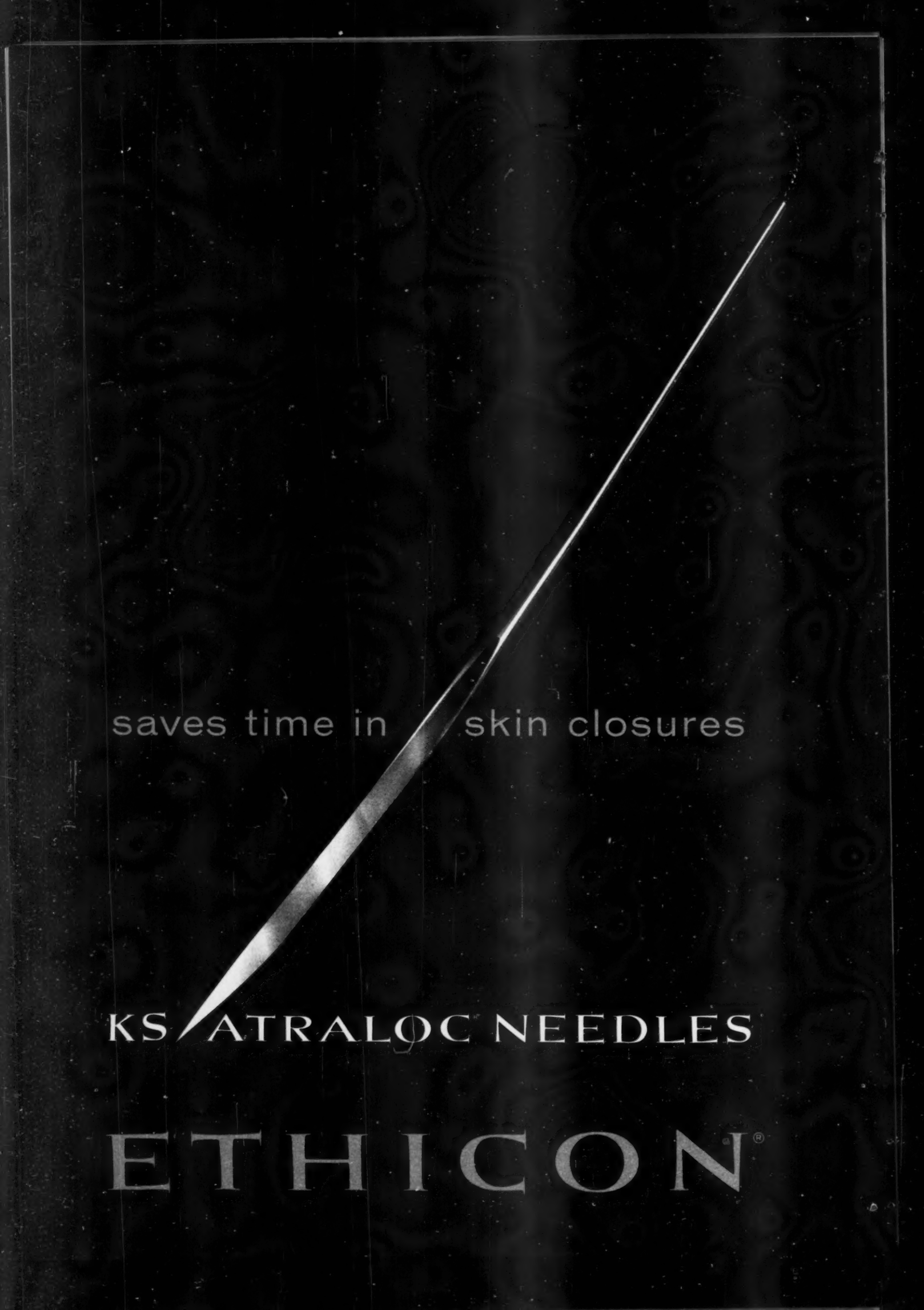
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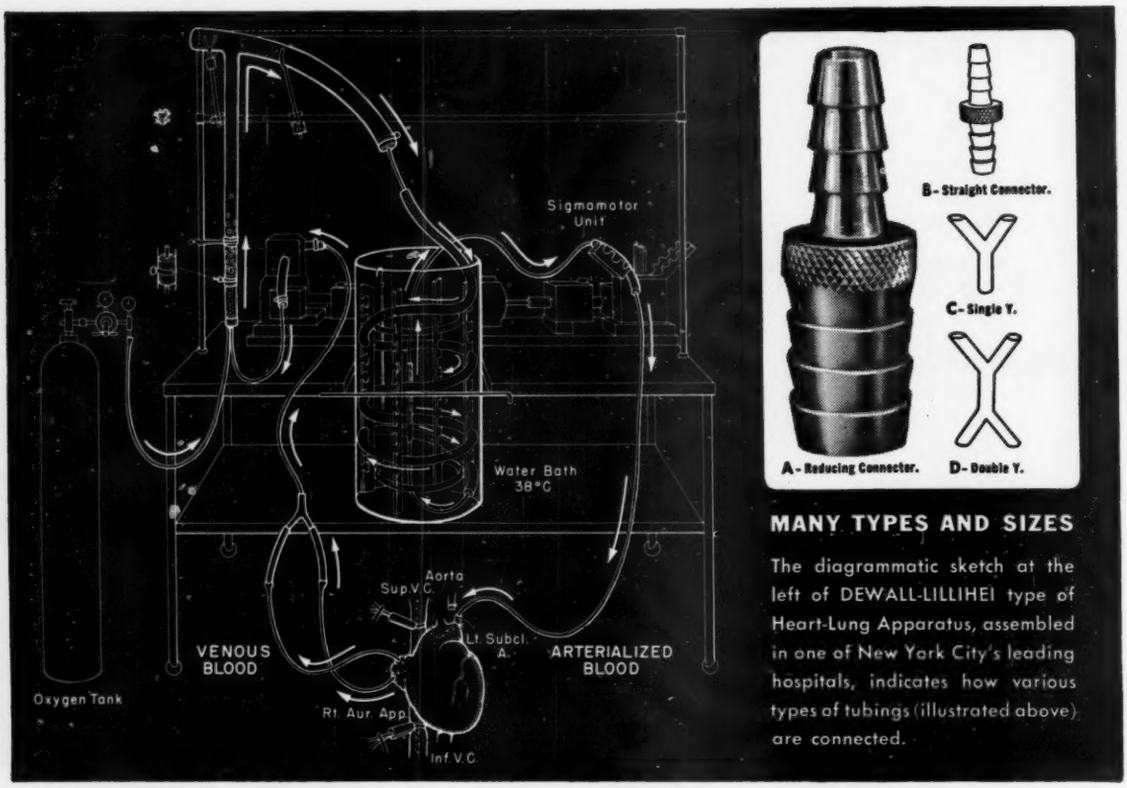

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The diagrammatic sketch at the left of DEWALL-LILLIHEI type of Heart-Lung Apparatus, assembled in one of New York City's leading hospitals, indicates how various types of tubings (illustrated above) are connected.

WECK Stainless Steel Connectors for Heart-Lung By-pass Systems

Hailed as one of the newest advances in Surgery, the "cardiac by-pass" operation, using artificial heart and lungs, has placed new demands on the manufacturers of special equipment and instruments.

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Also available is a complete assortment of Weck instruments for cardio-vascular surgery.

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January 4, 1957

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Sincerely yours,
Associate Attending Surgeon

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PANAFIL Ointment contains papain powder 10%, urea U.S.P. 10%, and water-soluble chlorophyll derivatives N.N.R. 0.5% in a hydrophylic ointment base.

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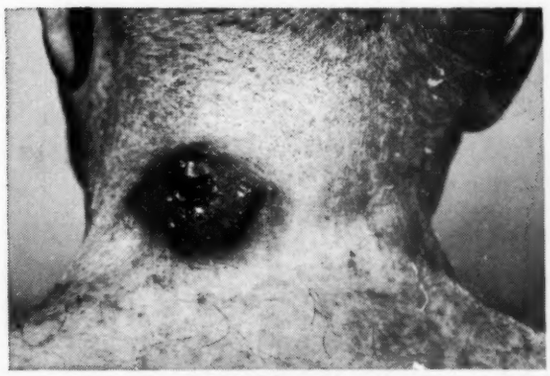
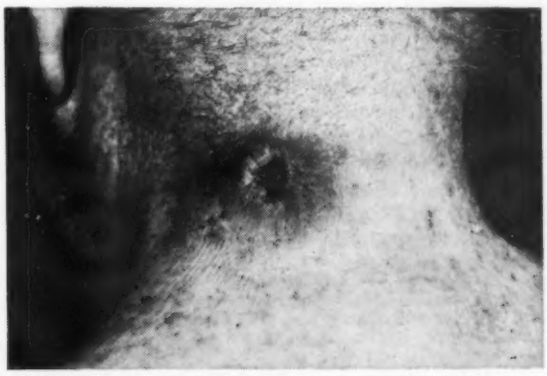
Severe carbuncle on back of neck of 79-year-old man.
Left: Before incision and start of therapy.
Right: Lesion almost healed after two weeks of therapy with PANAFIL. Complete healing one week later.





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References: (1) Miller, E. W.: New York J. Med. *56*:1446, 1956. (2) Gaines, A. L., and Barnard, R. D.: Angiology *8*:13, 1957. (3) Morrison, J. E., and Casali, J. L.: Am. J. Surg. *93*:446, 1957. (4) Wood, O. H.: Maryland M. J., in press.

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NEW INTRAMUSCULAR IRON PROVIDES PRECISION THERAPY, PROMPT RESPONSE

IMFERON,® the new intramuscular iron-dextran complex, was introduced to American hematologists at the Sixth International Congress of the International Society of Hematology held in Boston, August 27 to September 1, 1956. Recent experience from over 6 million injections has shown that this iron preparation is easy to administer, notably free from toxic effects, quickly absorbed and productive of rapid hematologic and clinical improvement. It has been termed "...the only therapeutically effective iron preparation for intramuscular use...."[1]

IMFERON meets the need for a safe, effective agent when parenteral iron is preferable for patients with iron deficiency anemia who are resistant or intolerant to oral iron, those with depleted iron reserves and those who require rapid restoration of hemoglobin, *e.g.* last trimester of pregnancy.

Previous parenteral iron preparations were unsatisfactory because of toxicity, pain on injection, or because they contained insufficient iron. IMFERON contains the equivalent of 5 per cent elemental iron. It is more stable than iron saccharate both *in vitro* and *in vivo* and does not precipitate in plasma over a wide pH range. It is isotonic with tissue fluids and has a pH of 5.2 to 6.0[1] Utilization for hemoglobin formation is almost quantitative.

Precision Therapy with IMFERON: Before treating a patient with IMFERON, total iron requirement is calculated by formula or determined from a convenient dosage chart. Then appropriate amounts of IMFERON are injected daily or every other day, until the total calculated required amount is given.

Iron Deficiency Anemia of Infancy: IMFERON provides a convenient safe means for restoring hemoglobin levels and iron reserves in anemic infants. Excellent results were obtained by Gaisford and Jennison[2] with IMFERON in 100 iron-deficient infants. From a pretreatment average of 54.5 per cent, hemoglobin levels rose to 87 per cent 10 weeks after the start of therapy. Clinical improvement paralleled this response. Premature infants and surgical cases were similarly benefited. IMFERON gave "...all the advantages of transfusion or intravenous therapy without the disadvantages."[2] There were no side effects in any of the infants treated. Wallerstein[3] confirmed these results, furnishing evidence that IMFERON is well absorbed and appears in the bone marrow 12 to 24 hours after injection. Results are equal to those with intravenous saccharated iron oxide without the unpleasant side effects. Sturgeon[4] showed that the first year's iron requirements in infancy can be supplied with three injections of IMFERON.

Iron Deficiency Anemia of Pregnancy: Nausea precludes oral iron therapy in many anemic pregnant women. In those with severe anemia who are first seen late in pregnancy, prompt hemoglobin regeneration is unobtainable with oral iron. IMFERON produced prompt hemoglobin responses in anemia of pregnancy,[5,6] the results being similar to those obtained with intravenous saccharated iron oxide. Side effects were virtually absent with IMFERON.[5,6]

Resistant Hypochromic Anemia: Patients who do not respond to oral iron, those who cannot take oral iron and those with gastrointestinal pathology respond well to injections of IMFERON.[7-11] While oral iron is of little value in treating the anemia of rheumatoid arthritis, IMFERON is "...as beneficial as intravenous iron and easier to administer."[8]

Present Studies: Published reports and recent findings of clinical investigators confirm the effectiveness and safety of IMFERON for hemoglobin regeneration and creation of iron stores. More than 70 studies are now being completed in the United States. Reports stress prompt hemoglobin response, ease of administration and freedom from side effects. Clinicians desiring additional information should request Brochure No. NDA 17, IMFERON, Lakeside Laboratories, Inc., Milwaukee 1, Wisconsin.

References: (1) Brown, E. B., and Moore, C. V., in Tocantins, L. M.: Progress in Hematology, New York, Grune & Stratton, Inc., 1956, vol. I, p. 25. (2) Gaisford, W., and Jennison, R. F.: Brit. M. J. 2:700 (Sept. 17) 1955. (3) Wallerstein, R. O.: J. Pediat. *49*:173, 1956. (4) Sturgeon, P.: Pediatrics *18*:267, 1956. (5) Jennison, R. F., and Ellis, H. R.: Lancet 2:1245 (Dec. 18) 1954. (6) Scott, J. M., and Govan, A. D. T.: Brit. M. J. 2:1257 (Nov. 27) 1954. (7) Grunberg, A., and Blair, J. L.: A.M.A. Arch. Int. Med. *96*:731, 1955. (8) Millard, J. B., and Barber, H. S.: Ann. Rheumat. Dis. *15*:51, 1956. (9) Baird, I. M., and Podmore, D. A.: Lancet 2:942 (Nov. 6) 1954. (10) Cappell, D. F.; Hutchinson, H. E.; Hendry, E. B., and Conway, H.: Brit. M. J. 2:1255 (Nov. 27) 1954. (11) Stevens, A. R.: A.M.A. Arch. Int. Med. *96*:550 1956.

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Reference: 1. Stafford, C. E., Kugel, A. I., and Dederer, A.: *Surg. Gynec. & Obst.* **89**:570, Nov.1949.

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1. Grayzel, H. G., and Schapiro, S.: Western J. Surg., Obstet. & Gynec., Oct. 1956.

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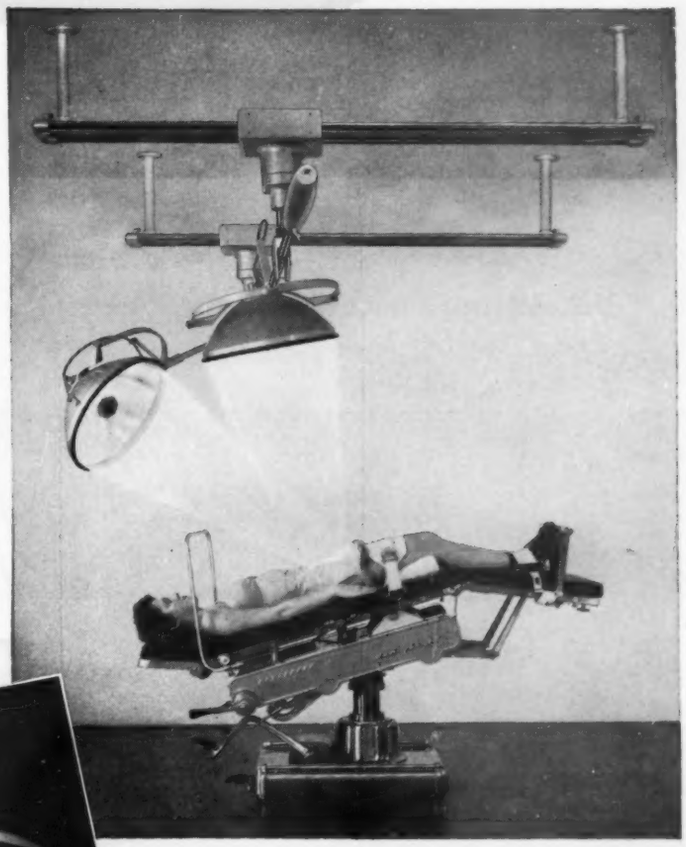

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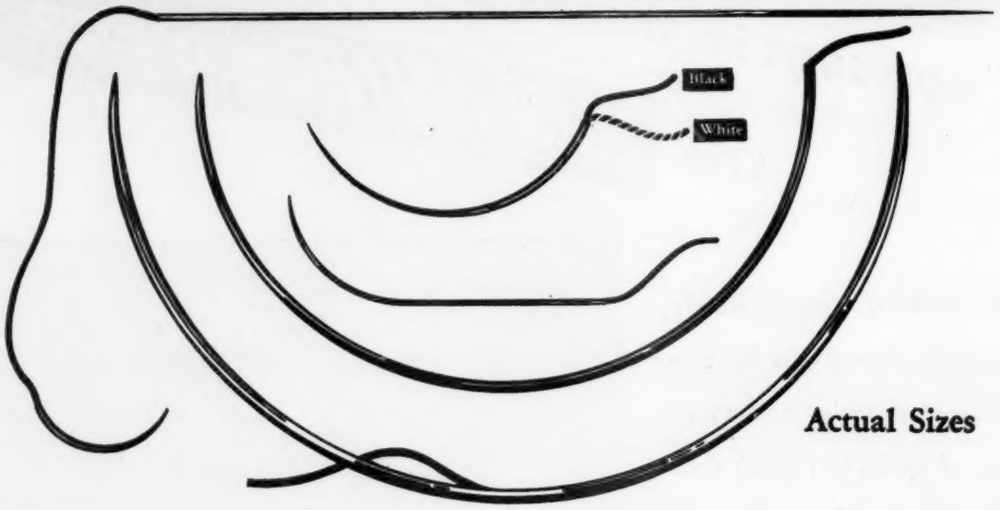

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new biliary abstergent

for **POSTOPERATIVE**

management of cholecystomized PATIENTS

*TRADEMARK OF G. D. SEARLE & CO.

ZANCHOL

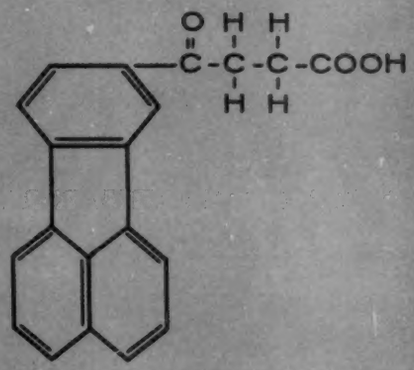
Zanchol—

bearing the generic name florantyrone, originated in the Searle Research Laboratories. It is unique in that it not only increases the volume of biliary flow but also changes the physicochemical properties of the bile. After Zanchol administration a greener bile[1] with little or no insoluble material is produced.

Zanchol has proved useful in preventing obstruction of T tubes and bile duct prostheses by sediment, and for the long-term management of patients whose gallbladders have been removed or for those who require reconstructive surgery of the common duct.

Chemistry

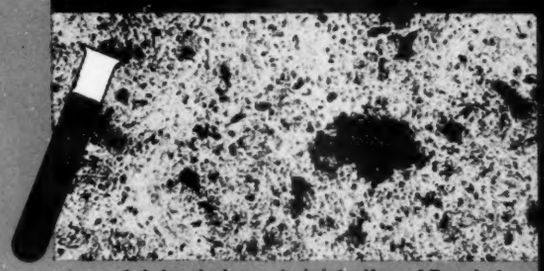
Zanchol is a synthetic fluoranthene derivative with a ketobutyric acid side chain. Unlike bile salts and bile acids it does not have a steroid structure. The formula is:



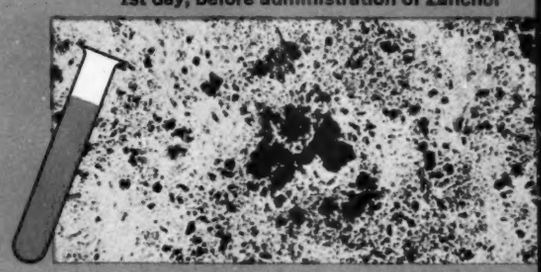
Improved abstergence and color change in bile produced by Zanchol. Tube No. 1, with light colored bile, considerable sediment and clouding, was from T-tube bile in a postcholecystectomized patient. Very little foam was obtained on shaking. Tube No. 2 contains bile from the same patient one day after the administration of Zanchol. The color is better, less sediment is present and the bile has greater clarity. The larger amount of foam obtained by shaking lasts for a longer period of time.

Photomicrographs[2]

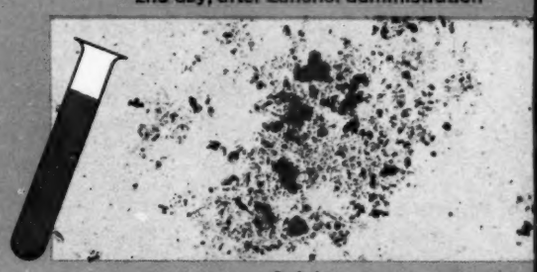
showing daily changes in sediment from centrifuged bile taken from T-tube drainage in a postcholecystectomized patient.



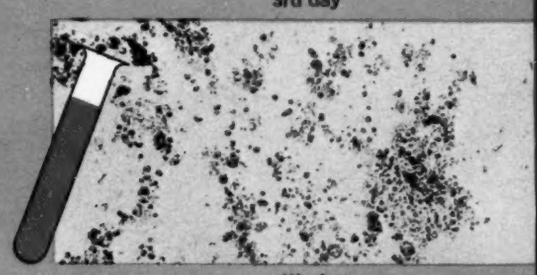
1st day, before administration of Zanchol



2nd day, after Zanchol administration



3rd day



4th day

5th day

new biliary abstergent

Clinical applications of Zanchol

The improvement in bile secretion provided by Zanchol indicates its use in the following conditions:

1. *After all biliary operations[1] to prevent the postcholecystectomy syndrome.*

   Routine use of Zanchol usually clears up the symptoms of eructation and abdominal pain or discomfort frequently occurring after cholecystectomy. Administration of Zanchol with meals provides an additional bile flow, thus partially compensating for the absence of a bile reservoir. Barowsky[2] observed that Zanchol often relieves postcholecystectomy distress of several years' duration.

2. *In postcholecystectomized patients with T-tube drainage.*

   Bilirubin sediment precipitates in T tubes and frequently obstructs bile flow. Removal of solids from the bile by the administration of Zanchol keeps T tubes free from sediment and encrustation. McGowan[1] states:

   > "After the first few patients had received this preparation, its usefulness was so obvious that it was prescribed routinely for all patients operated upon for gallbladder disease in whom it was necessary to place a T tube in the common bile duct for prolonged biliary drainage....
   >
   > "It has long been a known fact among doctors doing biliary surgery that tubes become covered and often plugged with orange colored sediment of bilirubin after a sojourn in the common duct and that a prosthesis, such as a rubber or metal tube, put into the duct to replace a destroyed portion of the duct invariably becomes plugged and reoperation is required. From this study, it is safe to conclude that such patients could be benefited indefinitely by the administration of SC-1674 [Zanchol]."

3. *In chronic cholecystitis when surgery is not employed.*

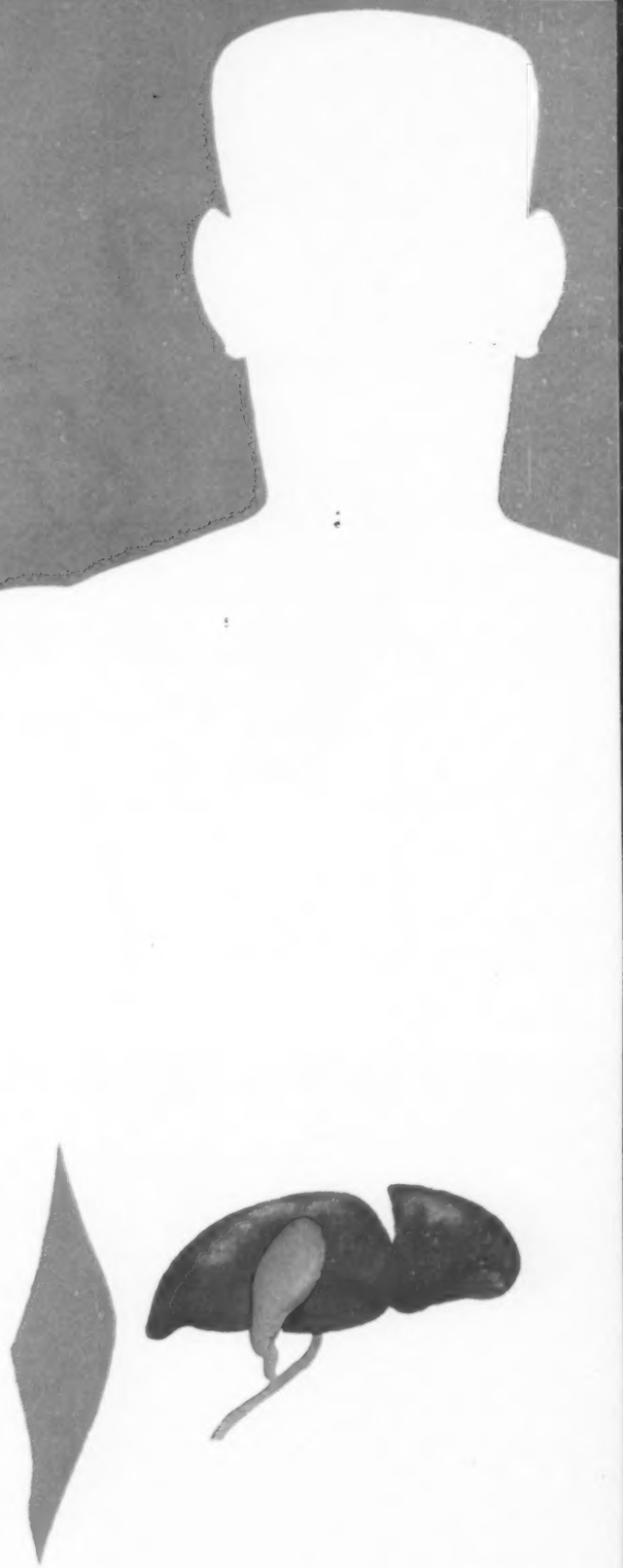
   Patients with chronic cholecystitis who are not considered good candidates for cholecystectomy will receive symptomatic relief from the administration of Zanchol.

The usual daily dosage is three or four tablets with meals and at bedtime.

Zanchol is available as tablets of 250 mg. each in bottles of 100 and 1,000.

1. McGowan, J. M.: Clinical Significance of Changes in Common Duct Bile Resulting from a New Synthetic Choleretic, Surg., Gynec. & Obst. 103:163 (Aug.) 1956.

2. Barowsky, H.: A Clinical Evaluation of the Choleretic Effect of a Synthetic Aromatic Keto Acid, SC-1674, to be published.



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ZANCHOL

Pharmacologic action in man

Direct observation[1] in cholecystectomized patients with T tubes in the common duct showed that Zanchol produced the following effects:

Bile Color Was Improved

In every patient the bile changed to a brilliant deep green, regardless of its original color.

Bile Sediment Disappeared

The sediment in biliary drainage fluid disappeared in five to ten days. Bilirubin and other slightly soluble material were dissolved.

Bacterial Content Decreased

After Zanchol was given there were no putrid odors in most bile specimens when allowed to stand at room temperature. Motile bacteria were reduced in number or were absent. However, since laboratory studies indicate that Zanchol has no bacteriostatic activity, this observation is probably the result of increased flow of bile and reduction of sediment and debris produced by Zanchol.

Volume and Fluidity Increased

A progressive increase in the amount of bile flow was noted. Viscosity was lowered and the bile appeared to flow more readily through the common duct.

Bile Abstergence Augmented

A definite abstergent action was observed in all patients. More foam was produced on shaking and the froth lasted for a longer period in bile specimens from Zanchol-treated patients.

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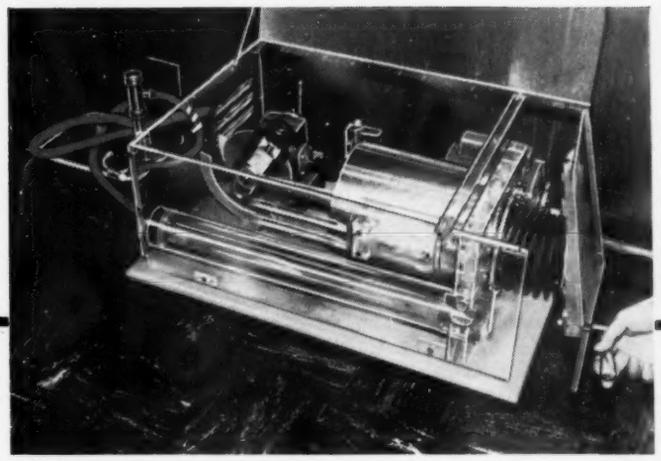

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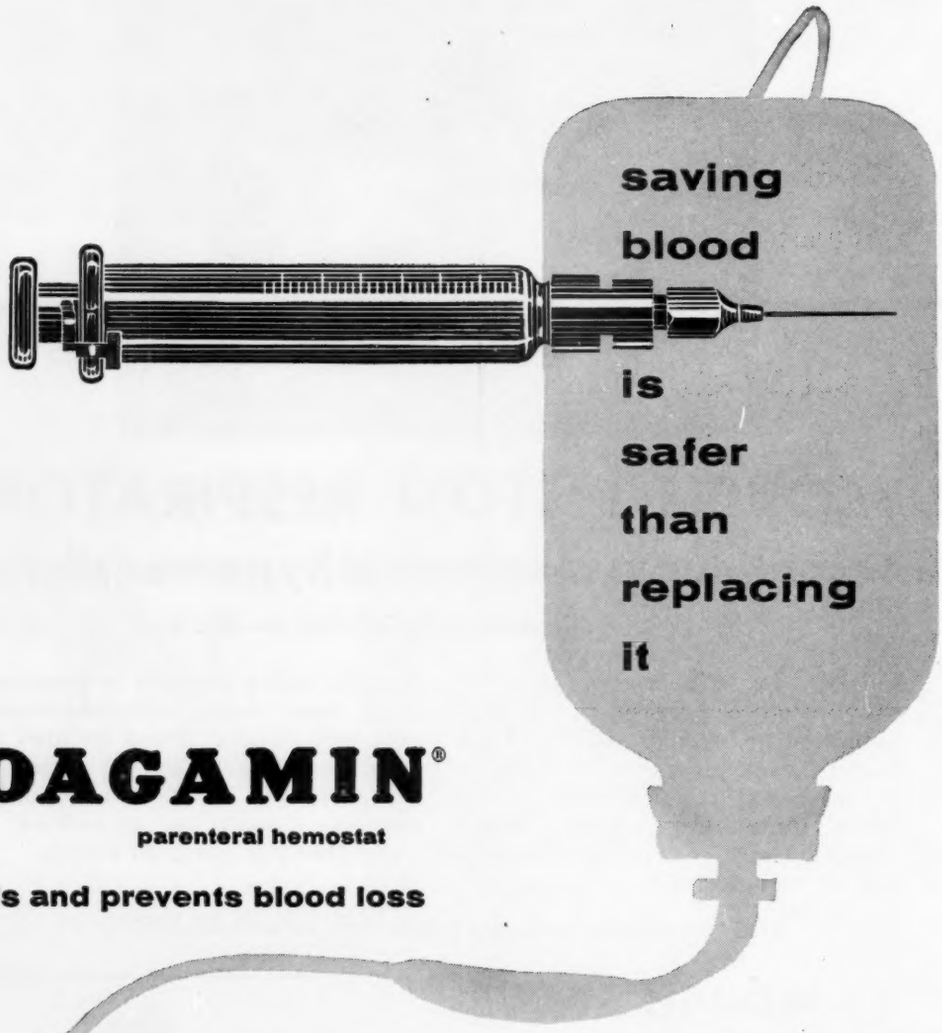

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1. Joseph, M.: Control of Hemorrhage—or Transfusion, Am. J. Surg. *87*:905, 1954. 2. Crisp, W. E.: Editorial; One Pint of Blood, Obst. & Gynec. *7*:216, 1956.

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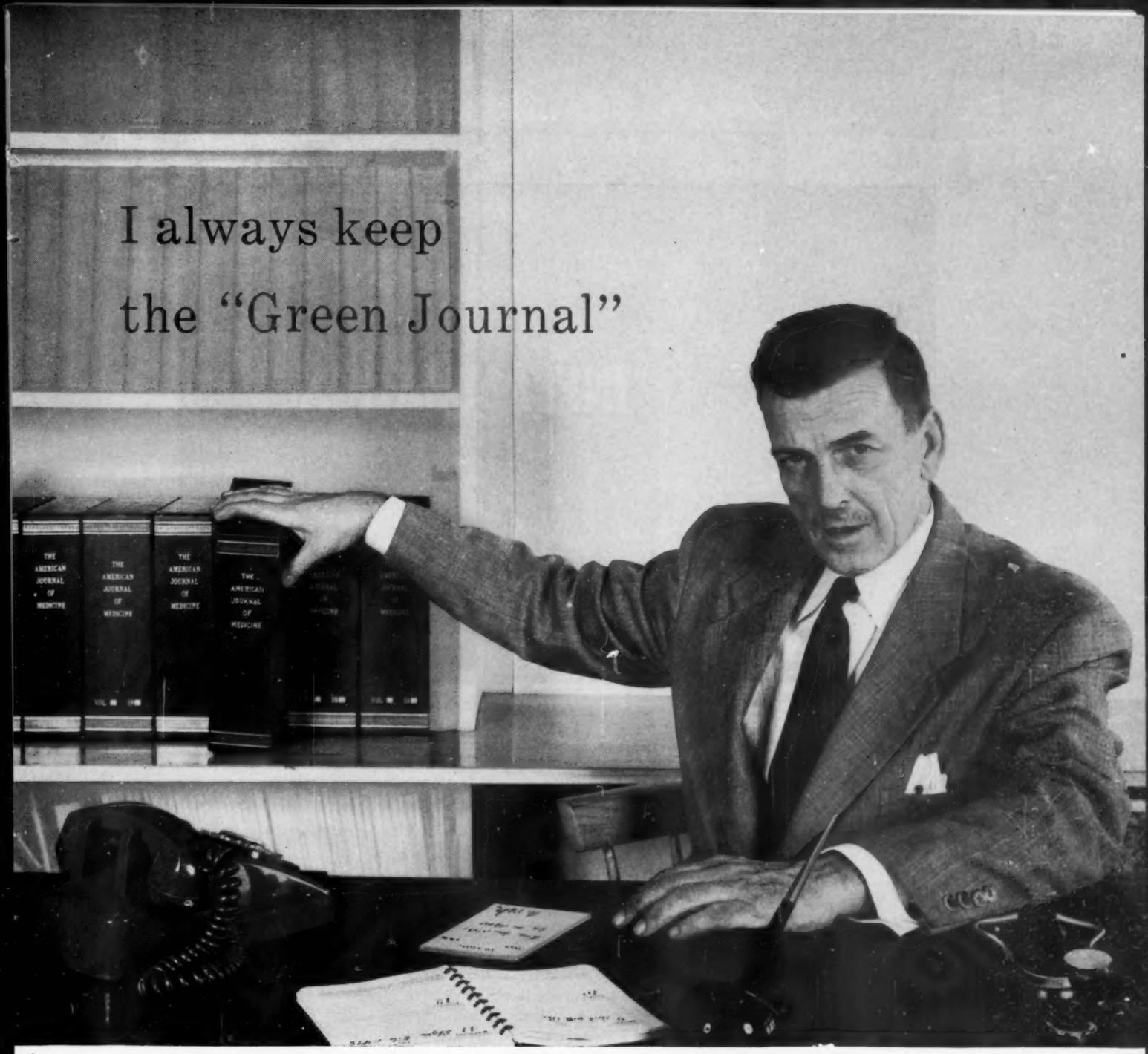

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1. Miller, J. M.; Surmonte, J. A.; Ginsberg, M., and Ablondi, F. B.: Postgraduate Medicine 20:260 (Sept.) 1956.

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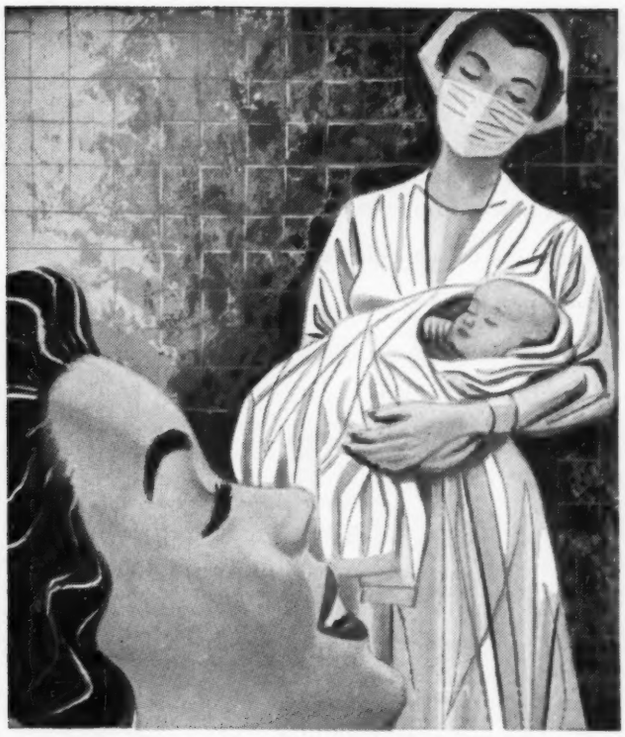

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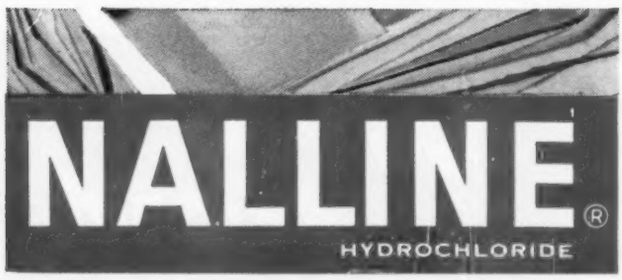

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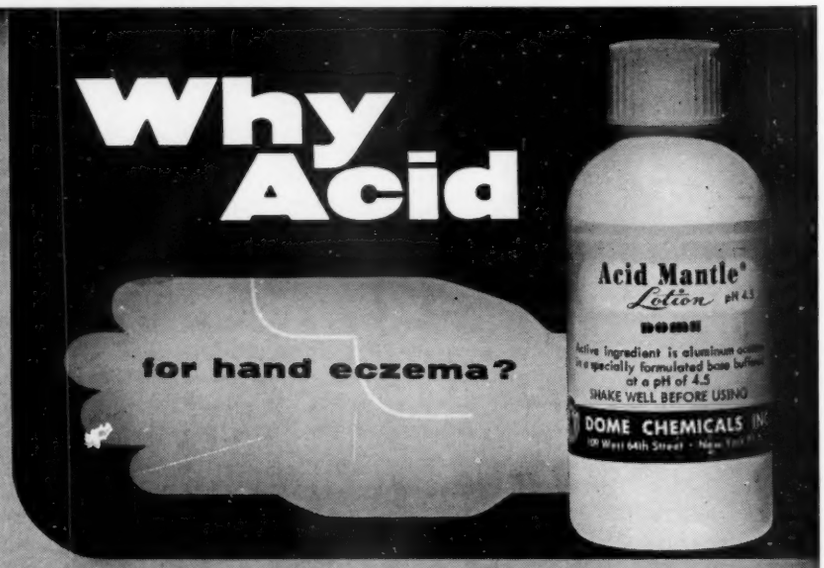

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REFERENCES: 1. Waisbren, B. A., and Crowley, W.: A.M.A. Arch. Int. M. 95:653, 1955. 2. Perry, R. E., Jr.: North Carolina M. J. 16:567, 1955.

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The American Journal of Surgery

VOLUME 93 JUNE 1957 NUMBER SIX

SYMPOSIUM ON MALIGNANT INTRACRANIAL NEOPLASMS

Introduction to Symposium

THE past few decades have witnessed a striking change in the attitude of the medical profession toward malignant disease. The uniformly dismal outlook for "cancer" has changed to one of hopefulness. The change has come about because of the progress which has been made in the treatment of malignant disease. This progress cannot be attributed to medicine alone, but has been achieved through advances in many scientific fields. Improvement in the treatment of other diseases stimulated physicians to assume a more vigorous approach to the treatment of cancer. The results of the vigorous treatment were so gratifying that it has been generally adopted and is now available for most types of malignancy in every part of the United States. With the improvement in the outlook for patients with cancer, physicians started to seek out malignancy instead of waiting for it to become obvious. The search for malignancy has led to safer and more accurate diagnostic technics, and the establishment of earlier treatment. The lay public has been educated to search for signs of early cancer in their own bodies, so that diagnostic tests and treatment can be started at the earliest possible moment. Early diagnosis and treatment is the best plan now available to the medical profession for the successful control of malignant disease.

During the same period of time knowledge of the central nervous system has increased, and diagnostic technics have been devised to equal or surpass those available for other parts of the body. The results of treatment of intracranial neoplasms have also improved to a comparable degree. Many physicians have assumed a more hopeful attitude toward the severe disability produced by brain tumors. However, there is a large group of physicians who still maintain the dismal attitude to intracranial neoplasms that was prevalent half a century ago. This dismal attitude arises in part from the complex nature of the brain, which makes the clinical diagnosis of brain tumors more difficult than that of tumors elsewhere in the body which show themselves by a mass, or through abnormal bleeding or discharge. The cardinal symptoms of other neoplasms are also produced by brain tumors, and can lead to an early diagnosis. The three papers in this Symposium which deal with the clinical diagnosis of intracranial neoplasms state repeatedly that pain in the form of headache, abnormal neuronal discharge in the form of convulsions, and abnormal function in the form of motor or sensory impairment are the characteristic signs of intracranial neoplasm. Masses which commonly give a clue to the hiding place of neoplasms are rarely present with a brain tumor, but the lack of this sign is made up for by disturbances in the control of the nervous system over the orderly function of the body.

In order to bring together present day knowledge of intracranial neoplasms in a concise form, the Indiana Division of the American Cancer Society invited eight outstanding men in the field to participate in their Ninth Annual Symposium on Malignancy. Each of the men

has contributed abundantly to the improvement in the outlook for patients with brain tumor. The first paper covers intracranial neoplasms from the point of view of the pathologist, bringing to the problem experimental as well as clinical experience. Primary tumors of the brain are discussed from a pathologic point of view because they assume the general pattern of malignancy seen in other parts of the body. In the section on clinical diagnosis, tumors which have a benign microscopic appearance are also discussed since they exert a "malignant" effect on the brain due to the pressure which the neoplasm exerts within the rigid confines of the skull. The greatest portion of the Symposium is devoted to clinical diagnosis because this is the area which permits early detection. An arbitrary division of intracranial neoplasms has been made for the discussion of clinical diagnosis to emphasize the differences between the signs and symptoms in children and adults, and also the similarities and differences between neoplasms above and below the tentorium cerebelli. Diagnostic aids have assumed a very important role in the handling of intracranial neoplasms. Although x-ray contrast studies of the cerebral ventricles and blood vessels have developed into almost essential elements of the diagnostic work-up of intracranial neoplasms, these are tools of the specialist and usually are not available to the physician who first sees a patient with suspected brain tumor. For this reason the discussion on x-ray emphasizes the changes which intracranial neoplasms make in the plain skull films. Although electroencephalography and radioisotopes do not as yet give the accurate localization of intracranial neoplasms achieved by x-ray contrast studies, they are newer diagnostic methods which show a great deal of promise. They are useful for screening, to aid in determining which patients should be subjected to further diagnostic testing. The surgical technic for dealing with intracranial neoplasms has improved to such an extent that it acts as a stimulus to neuropathologists, clinical diagnosticians, neuroradiologists and electroencephalographers. No discussion of intracranial neoplasms would be complete without recognizing this outstanding medical advance.

The striking progress in the diagnosis and treatment of intracranial neoplasms outlined in this Symposium is to a great extent due to the function of specialists in the field of the nervous system. Yet the fact that this branch of medicine has become so specialized does not free the pediatrician, the internist, the general practitioner or other physicians from their obligation to the patient with an intracranial neoplasm. On the contrary, it increases their obligation to become suspicious of an intracranial neoplasm in time to save the patient severe disability.

I wish to express my gratitude to the eight authors for their cooperation in the preparation of their manuscripts, and to Drs. Orville T. Bailey, Alex T. Ross, John R. Russell and L. W. Freeman, the Neurological Surgery House Staff and the members of the cancer committee for their help in the planning and execution of the program.

ROBERT F. HEIMBURGER, M.D.
Indianapolis, Indiana

The Natural History of Intracranial Neoplasms, with Special Reference to the Gliomas*

HARRY M. ZIMMERMAN, M.D., *New York, New York*

From the Laboratory Division, Montefiore Hospital, New York, New York.

IT is variously estimated that primary intracranial neoplasms represent from 2 to 5 per cent of all tumors of the human body. Of these intracranial tumors, slightly less than one-half belong to the glioma group. The next most common tumor in this location is the meningioma, with an incidence of from 10 to 15 per cent. Pituitary tumors are next with a probable incidence of from 5 to 10 per cent, and these new growths are followed by a 3 to 8 per cent incidence for the neurinomas.

Together, the congenital tumors within the skull form 5 to 7 per cent of the total and consist of the craniopharyngiomas, cholesteatomas, certain pinealomas, chordomas, hemangiomas, colloid cysts and lipomas. Intracranial sarcomas, including those of leptomeningeal as well as perivascular origin and also inclusive of the cerebellar type, represent about 3 per cent of all intracranial neoplasms.

The only remaining large group of intracranial tumors comprises the metastatic neoplasms. Here it should be noted that statistics based on necropsy material contain an appreciably greater number of metastatic tumors than do those derived from biopsied cases. This is because it is still common practice in neurosurgery to avoid operating on patients known to have metastatic disease. In Table 1 a summary of the incidence of the more common intracranial tumors is presented [1].

RADIOSENSITIVITY

In 1921, Ewing [2] postulated the response of various types of brain tumors to radiation. By his criteria, tumors should be highly sensitive if they are richly cellular, have many undifferentiated cells, contain numerous mitoses, are highly vascularized with delicate capillaries, and contain little intercellular substance or stroma. Conversely, tumors should be insensitive if they grow slowly, are composed of adult, well differentiated cells with few mitoses, and have a blood supply of adult vessels and a rich intercellular stroma.

Were these criteria completely valid, the

TABLE 1
INTRACRANIAL TUMORS

Tumors	New Haven Hospital Series		Cushing Series*	
	No. (758)	Per cent	No. (2,209)	Per cent
Gliomas	335	44.3	944	42.7
Meningiomas	82	10.8	302	13.7
Neurinomas	20	2.6	191	8.6
Pituitary tumors	30	3.9	395	17.9
Congenital tumors:				
Craniopharyngiomas	16	2.1	95	4.3
Cholesteatomas	2	....	13	0.6
Pinealomas	6	....	16†	0.7
Chordomas	1	....	2	....
Hemangiomas	19	2.5	45	2.0
Colloid cysts	5	....	...	....
Lipomas	3	....	...	....
Sarcomas	27	3.5	13	0.6
Papillomas of choroid plexus	1	....	12	0 6
Metastatic tumors	102	13.3	93	4.2
Granulomas:				
Abscesses	70	9.1	...	....
Tuberculomas	36	4.7	33	1.5
Gummas	3	....	12	0.5
Miscellaneous	...	....	59	2.7

* Cited by Eisenhardt, L. in Proceedings of the National Cancer Conference, 1949, pp. 227–228.

† In the Cushing series the pinealomas are included among the gliogenous neoplasms numbering 944.

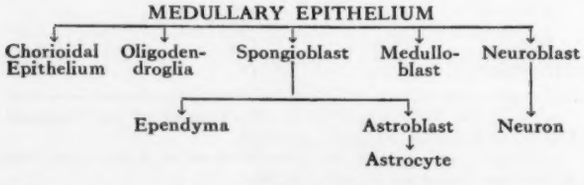
* Read at the Ninth Annual Symposium on Malignancy given at the Indiana University Medical Center, Indianapolis, Indiana.

glioblastoma multiforme should be the most sensitive to radiation. Experience has shown, however, that radiotherapy applied to this tumor has in general been disappointingly ineffective. On the other hand, the medulloblastoma which, like the glioblastoma multiforme also meets Ewing's criteria for roentgen sensitivity, is indeed remarkably responsive to radiation. Tumors like the pituitary adenomas, craniopharyngiomas and meningiomas, which should be non-responsive to radiation according to Ewing's postulates, have frequently shown surprisingly good responses. The pituitary tumors have especially been favorably influenced by this form of therapy. There is also evidence to indicate that some of the less malignant gliomas, such as the ependymomas, astrocytomas and oligodendrogliomas, have been arrested in growth with consequent prolongation of life in response to roentgen rays and other forms of radiation. These results would seem to indicate that, lacking other means of treatment in a given case, radiation therapy may be tried with due awareness of the danger in such therapy to neural and other tissues.

THE GLIOMAS

Of all the intracranial neoplasms, those of the glioma group are much the most important. This is not only because of their numerical preponderance but also because of the following considerations: (1) their histiogenesis is still debatable; (2) their classification is often difficult and on occasion arbitrary; (3) their sites of predilection in different parts of the brain seem capricious; and (4) their unalterable failure spontaneously to metastasize distantly, that is, extracranially, offers a unique example in the behavior of malignant neoplasms.

Histiogenesis. It is generally accepted that the different glial cells of the mature nervous system, with the possible single exception of the microglia, are all derived from embryonal neurectoderm.



This origin is shared by the glia and neurocytes alike. Furthermore, the primitive neurectoderm or medullary epithelium differentiates by recognizable stages to the fully mature glia.

The adult gliacyte may give rise, under certain conditions, to a glial neoplasm by proliferation. It is assumed that a *single* adult glial cell is stimulated to proliferative activity and that from this single cell a tumor is produced. To account for the common observation that in any given glioma there are present tumor cells of variable degrees of maturity and even of different types, another theory has been elaborated. This is the theory of dedifferentiation. It is widely accepted and is regarded by many as a sort of biologic law.

Dedifferentiation is held to account for the appearance of pleomorphism which any given glioma may present. But does it account for the presence in a given tumor of clearly recognizable neoplastic gliacytes of different *types*, such as astrocytes in an ependymoma, or spongioblasts in an oligodendroglioma? To emphasize the frequency of occurrence of glial cells of different types in a single glioma and to give some indication of the variety of such cells, the following cases are cited.

The first patient was a young man who had a brain tumor removed in September, 1949. The tumor involved the brain stem, more especially the medulla oblongata, but a sufficient sample was removed for microscopic study. (Fig. 1.) It revealed a typical ependymoma with perivascular rosettes. The patient made a surprisingly good recovery from the operation and was then treated with radiation. He returned to work and was in relatively good health for about two years. He then began to fail and died in June, 1953. Necropsy revealed that remnants of the original tumor were still present in and around the medulla, but much of the neoplasm was now composed of strikingly pleomorphic cells, giant tumor cells and spongioblasts. (Fig. 2.) The tumor was no longer, if indeed it ever was, a "pure" or uniform ependymoma. Postmortem examination revealed a "mixed" glioma, in part an ependymoma and in part a glioblastoma multiforme.

The second patient was a fifty-three year old man who was found to have a cystic astrocytoma in the frontal lobe on exploratory craniotomy. (Fig. 3.) This tumor had the usual benign appearance and did not presage the course the patient was to follow. Within a

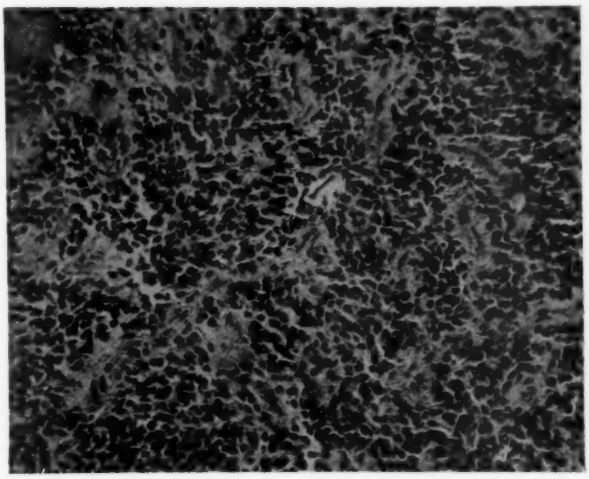


FIG. 1. Ependymoma involving medulla oblongata. Note perivascular rosette at top center of photomicrograph. Hematoxylin and eosin stain, original magnification ×150.

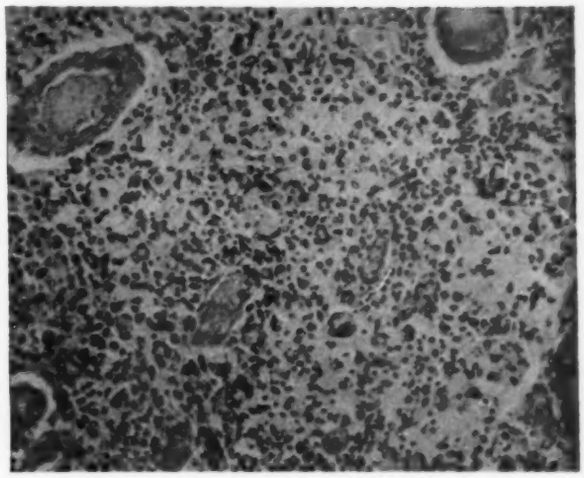


FIG. 2. Later appearance of neoplasm from same patient whose first biopsy specimen is shown in Figure 1. Tumor is now pleomorphic and contains multinucleated tumor giant cells. Hematoxylin and eosin stain, original magnification ×150.

year the tumor recurred and required reoperation. Once again it was an astrocytoma but no longer cystic, and it was considerably more cellular. (Fig. 4.) Calcium salt deposits were present and were in keeping with a slowly growing astrocytoma. Within seven months, however, the patient had returned to the hospital where, after a stormy course of less than four months, he died. At necropsy a huge tumor was found replacing much of the right cerebral hemisphere, infiltrating the basal ganglions and extending into the left hemisphere by way of the corpus callosum. On microscopic study, the neoplasm (Fig. 5) contained large zones of necrosis with spongioblasts forming the typical pseudopalisades of a glioblastoma multiforme. Also noted were the thrombosed blood vessels and the multinucleated tumor giant cells characteristic of this neoplasm. Parts of the very large tumor still consisted of astrocytoma.

The third and final illustrative case is presented through the kindness of Dr. Alfred Angrist. The patient was a forty year old woman who in retrospect had symptoms of an expanding intracranial lesion for one year prior to death. A large tumor was found in the frontoparietal region on postmortem examination, and this neoplasm was in part cystic (Fig. 6) and contained small quantities of thick, amber-colored fluid. On microscopic study, this portion of the tumor had the classic appearance of a cystic astrocytoma. A major portion of the neoplasm, however, had a completely different appearance. (Fig. 7.) It was firm, non-cystic and contained large amounts of calcium salt deposits. Microscopically, the presence of calcium was confirmed and, in addition, it was discovered that this portion of the tumor was composed entirely of oligodendroglia.

To these examples of gliomas which are "mixed" tumors at the start and which apparently become transformed from one type to another spontaneously, or following operative intervention or radiotherapy, may be added many more. Almost every glioma contains, in addition to the distinctive cells from which its name is derived, variable numbers of other glial elements. Classification of a gliogenous neoplasm is thus based upon the identification of the most malignant cells present and/or the predominance of one kind of cell. Utilizing these principles of classification, the incidence of intracranial gliomas [*1*] is presented in summary in Table II.

Terminology and Classification of Gliomas. The obvious disadvantages of the classification presented, which was evolved from near chaos by Bailey and Cushing in 1926 [*3*], are the rather large number of individual tumors included and, as already indicated, the fact that certain gliomas are derivatives of more than one basic glial type. The latter is meant to indicate that certain gliomas are of "mixed"

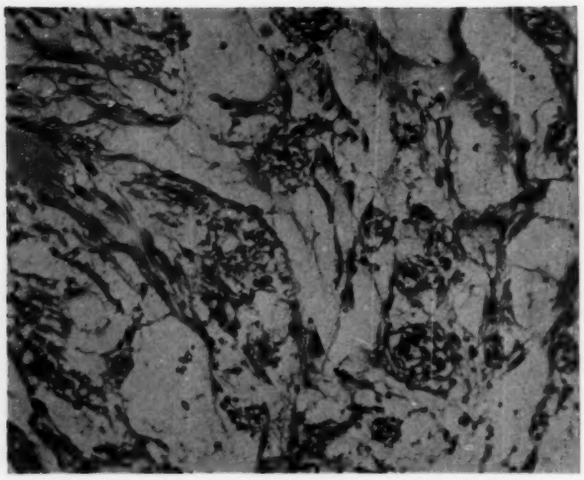


FIG. 3. Cystic astrocytoma in frontal lobe of a fifty-three year old man. Hematoxylin and eosin stain, original magnification ×150.

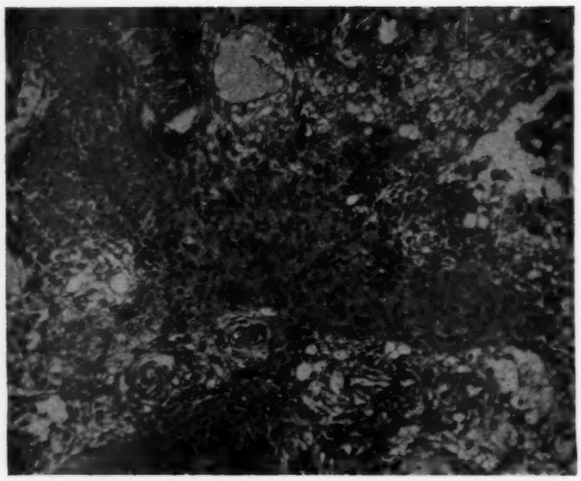


FIG. 4. Photomicrograph of cellular astrocytoma removed one year later from patient described in Figure 3. Hematoxylin and eosin stain, original magnification ×150.

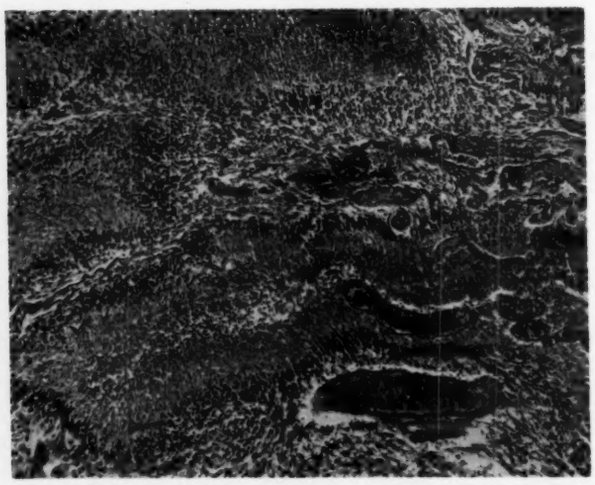


FIG. 5. Microscopic appearance of neoplasm in right cerebral hemisphere removed eleven months later from patient described in Figures 3 and 4. Pseudopalisading of spongioblasts is apparent. Hematoxylin and eosin stain, original magnification ×150.

origin and defy classification. Efforts have been made from time to time to simplify the terminology, and a recent attempt of this kind was made by Kernohan, Mabon, Svien and Adson [4]. These workers suggested a classification which is based on the idea that gliomas arise from the adult astrocyte, oligodendrocyte and ependymal cell. This concept provides three basic glial neoplasms, namely, astrocytoma, oligodendroglioma and ependymoma. Kernohan and his co-workers assume that more malignant neoplasms develop from these basic types by the process of dedifferentiation. For example, in their classification the glioblastoma multiforme is an astrocytoma of grade III or IV malignancy.

There are at least four objections to this approach in classification of the gliomas. One is that in reality it is no simpler than the classification adopted by Bailey and Cushing. If four numerical designations for each of the three basic types of glioma are employed, as proposed by Kernohan and his associates to designate morphologically distinct tumors, there are still twelve gliogenous neoplasms to be diagnosed. Another objection is the one inherent in all classifications which resort to

TABLE II
INTRACRANIAL GLIOMAS

Gliomas	New Haven Hospital Series		Cushing Series*	
	No. (335)	Per cent	No. (944)	Per cent†
Astrocytomas	83	24.7	274	29.0
Astroblastomas	8	2.4	46	4.9
Ependymomas	13	3.9	29	3.1
Glioblastomas multiformes	161	48.0	242	25.6
Medulloblastomas	32	9.6	90	9.5
Oligodendrogliomas	18	5.4	38	4.0
Spongioblastomas polares	7	2.1	35	3.7
Unclassified gliomas	13	3.9	167	17.7

* Cited by Eisenhardt, L. in Proceedings of the National Cancer Conference, 1949, pp. 227–228.

† The total percentage of gliogenous neoplasms in Cushing's series is 97.5 because the medulloepitheliomas, neuroepitheliomas, ganglioneuromas and pinealomas are not included.

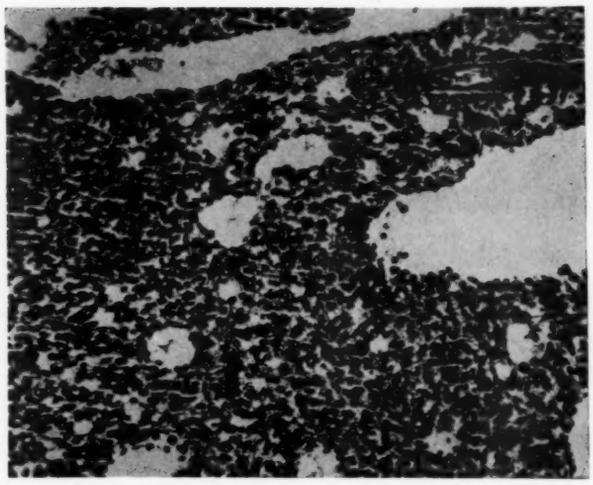


FIG. 6. Photomicrograph of cystic astrocytoma in frontoparietal region of a forty year old woman. Hematoxylin and eosin stain, original magnification ×150.

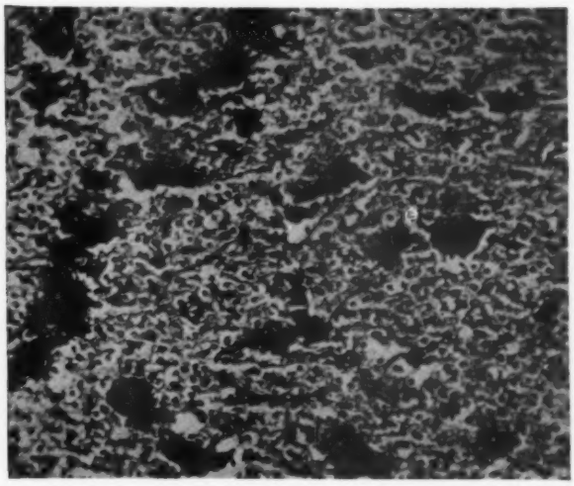


FIG. 7. Oligodendrogliomatous portion of neoplasm, with calcification, found in patient described in Figure 6. Hematoxylin and eosin, original magnification ×150.

numerical grading of degrees of malignancy, since it implies a prophetic capacity on the part of the pathologist. Still another objection is the failure of the classification to provide for the medulloblastoma in a scheme of tumors originating from the three adult glial cell types. But perhaps the major objection to the scheme is the assumption that each glioma takes origin from a *single* cell, such as an astrocyte, and that the developing tumor is merely the result of proliferative activity on the part of that glial element. Actually, as will be shown presently through the results of experiments, many if not all gliomas contain mixtures of different proliferating gliacytes.

Experimental Gliomas. For more than fifteen years my associates, Drs. Arnold, Maier, Netsky and Terry, and I have produced gliogenous neoplasms in several inbred strains of mice by the implantation of minute pellets of chemical carcinogens (methylcholanthrene, benzpyrene and dibenzanthracene) in various locations within the brains of these animals. In this way it has been possible to demonstrate that the type of glioma induced with carcinogen implantation varied in large measure with the locations in which the pellets were placed [5–7]. Pellets in contact with the ventricular ependyma induced the formation of ependymoma or ependymoblastoma (Fig. 8); those in the subcortical white matter often induced glioblastoma multiforme (Fig. 9) and, far less frequently, astrocytoma; in the occipital lobes the usual tumor was the oligodendroglioma (Fig. 10); in the corpus callosum it was the spongioblastoma polare (Fig. 11); in the cerebellum the induced tumor was most often the medulloblastoma. (Fig. 12.)

It soon became apparent, however, that very few of the experimental neoplasms were "pure" tumors, that is, composed of a single cell type. Most of the tumors, rather, were composed of several different cell types even though the preponderance of neoplastic cells of one variety justified the appellation or classification chosen for the tumor as a whole. In other words, most of the gliomas were "mixed" tumors.

In 1940 Dr. Arnold and I produced a "mixed" or "multiple" glioma in the brain of a mouse in which we implanted a pellet of methylcholanthrene [5]. Part of the neoplasm was composed of spongioblasts which formed pseudopalisades on the edges of necrotic foci as seen in glioblastoma multiforme (Fig. 13), and part was composed of an oligodendroglioma. (Fig. 14.) On transplantation subcutaneously into animals of the same mouse strain, the glioblastomatous portion of the primary cerebral tumor continued reproducing a glioblastoma in generation after generation, and the parallel series of subcutaneous transplants from the oligodendrogliomatous portion continued to reproduce oligodendrogliomas. Commenting on this phenomenon in 1941, we wrote in *Cancer Research* [5], "Such results are comparable to the everyday experience of the

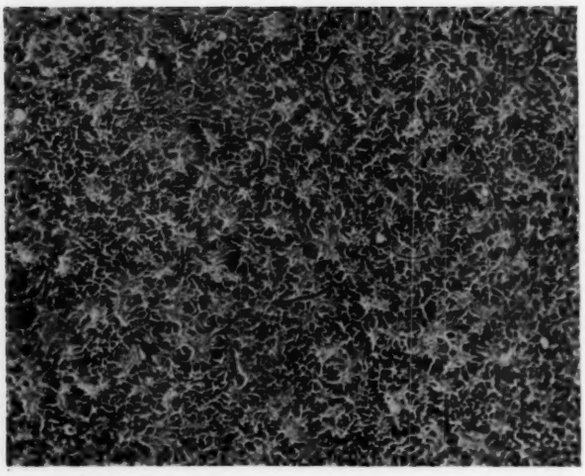


FIG. 8. Photomicrograph of ependymoma produced by action of methylcholanthrene on ventricular ependyma. Hematoxylin and eosin stain, original magnification ×150.

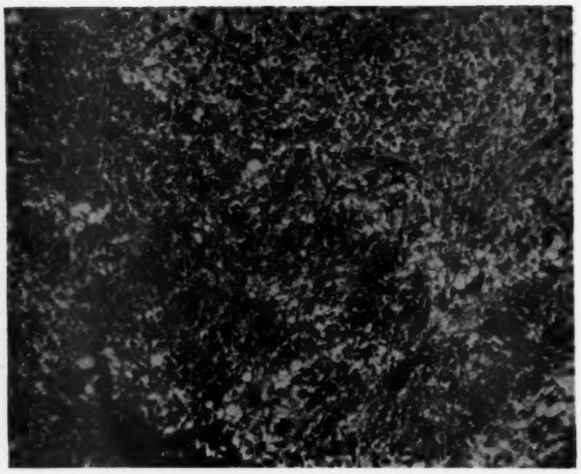


FIG. 9. Glioma produced with chemical carcinogen in subcortex of parietal lobe. Most of the cells are spongioblasts arranged in pseudopalisades around cores of necrosis, as seen in glioblastoma multiforme. Note oligodendrocytes, however, in top center of photomicrograph. Hematoxylin and eosin stain, original magnification ×150.

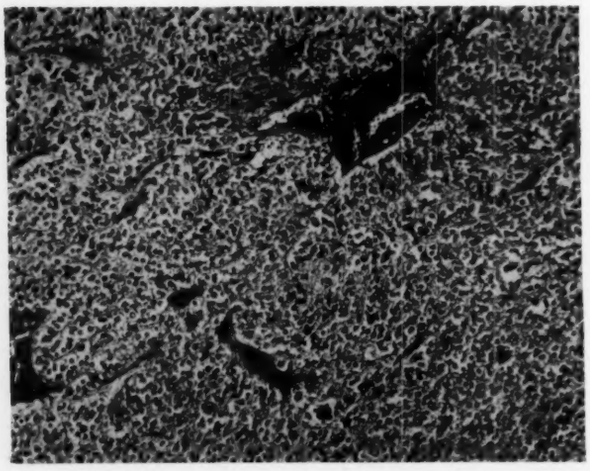


FIG. 10. Carcinogen-induced oligodendroglioma. Pellet of methylcholanthrene deposited in white matter of occipital lobe. Hematoxylin and eosin stain, original magnification ×150.

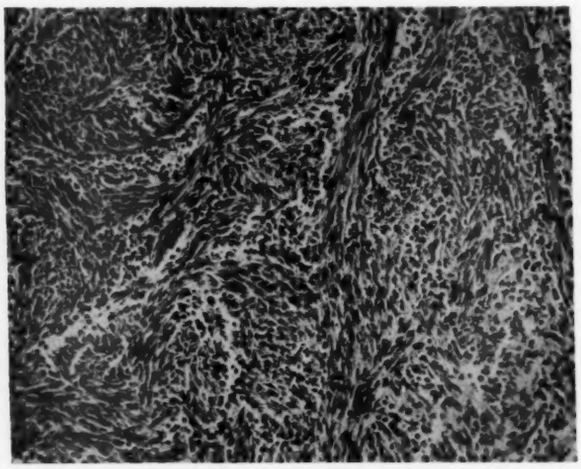


FIG. 11. Polar spongioblastoma produced with methylcholanthrene in corpus callosum. Note characteristic interlacing bands of spongioblasts. Hematoxylin and eosin stain, original magnification ×150.

bacteriologist who subcultures colonies from a mixed bacterial growth to obtain several pure strains of organisms."

In a more recent experiment, Dr. Maier and I produced a primary "mixed" cerebral glioma with methylcholanthrene. On serial transplantation, this tumor yielded a typical glioblastoma (Fig. 15) with necrotic zones and marginal arrangement of spongioblasts in pseudopalisades. Another fragment of the same primary tumor, on the other hand, produced large but slowly growing cystic tumors in subcutaneous transplants. These contained large quantities of amber-colored fluid that clotted on standing. (Fig. 16.) Such fluid, of course, is often present in certain cystic astrocytomas in man. On microscopic study, this transplanted glioma did indeed reveal the loose fibrillary and cystic structure of an astrocytoma. (Fig. 17.) Parallel transplants of the two distinctly different tumors derived from but a single primary cerebral neoplasm each maintained its distinct morphologic identity for more than thirty generations to the conclusion of the experiment.

Scrutiny of the phenomena which attend the

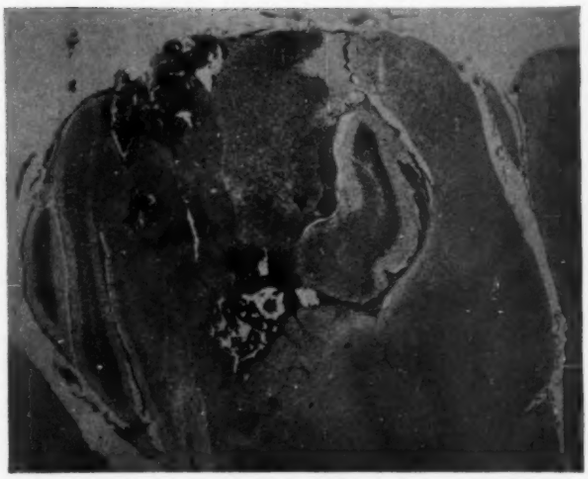


FIG. 12A. Photomicrograph of glioma in cerebellum at site of carcinogen implantation. Hematoxylin and eosin stain, original magnification ×24.

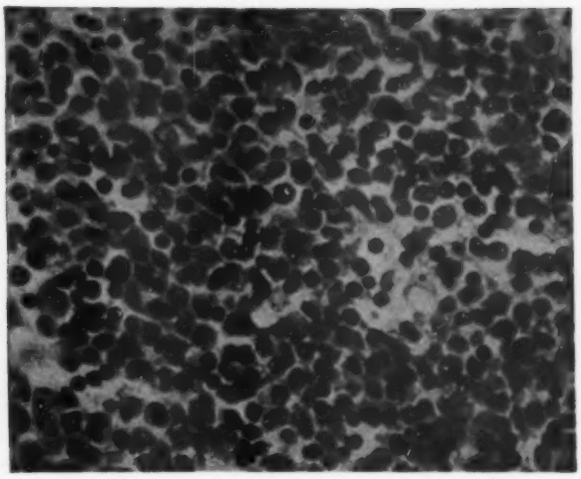


FIG. 12B. Higher magnification of medulloblasts composing chemical-induced tumor shown in Figure 12A. Note almost naked nuclei of tumor cells and scant stroma. Hematoxylin and eosin stain, original magnification ×545.

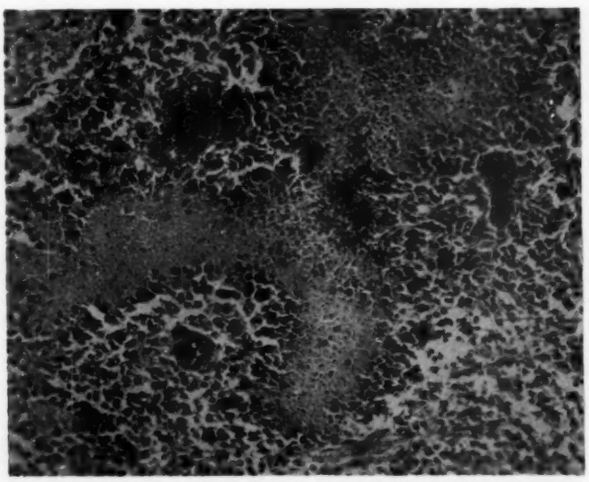


FIG. 13. Photomicrograph of part of experimentally induced "mixed" glioma. This part contains the necrosis, spongioblasts and multinucleated tumor cells of a glioblastoma multiforme. Hematoxylin and eosin stain, original magnification ×150.

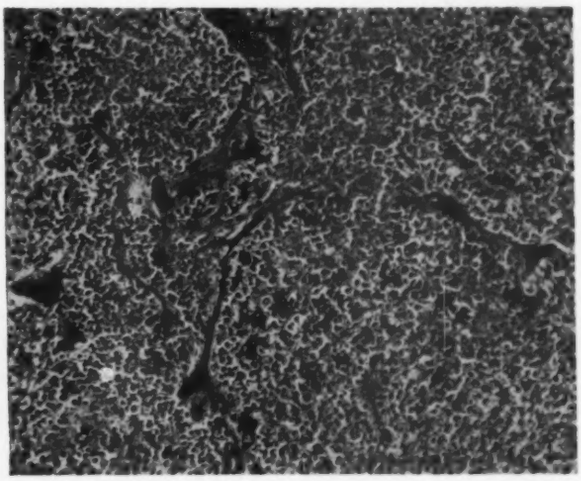


FIG. 14. Photomicrograph of another part of the same experimentally induced "mixed" glioma shown in Figure 13. Here the cells are all oligodendrocytes with halos around centrally situated nuclei. Hematoxylin and eosin stain, original magnification ×150.

origin of the carcinogen-induced experimental brain tumors is rewarding from the standpoint of the histiogenesis of these neoplasms. Following the implantation of the chemical in the animal's brain, a long quiescent period ensues which lasts well over 200 days. Only then does the animal which is destined to develop a brain tumor—this occurs in about 50 per cent of the animals—exhibit any manifestations of a space-occupying lesion. At about this time or shortly before, the zone immediately adjacent to the space occupied by the pellet of carcinogen shows evidence of some cellular activity. In the cerebellum, for example, one or two multinucleated giant cells, a very slight amount of nuclear debris and a trifling glial proliferation may be seen. (Fig. 18.) In another animal at about this stage, with the carcinogen in the lateral ventricle, an incipient glioma has made its appearance. (Fig. 19.) This early lesion already shows some injury to, and perhaps also some proliferation of, the ependyma, but in addition there is a lively subependymal glial response in which a number of cells

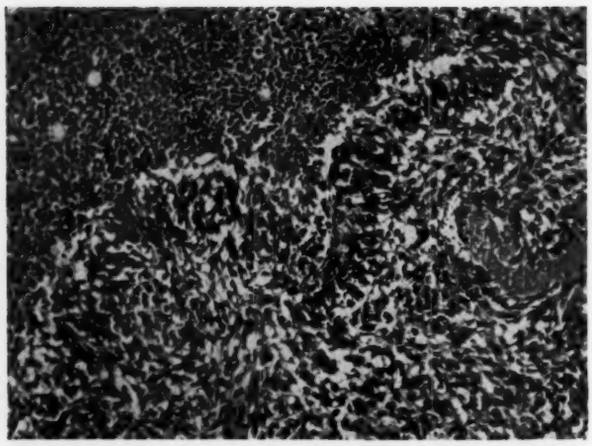


FIG. 15. Appearance of spongioblasts arranged in pseudopalisades on margin of necrosis in subcutaneous transplant from a primary "mixed" cerebral glioma produced with methylcholanthrene. Hematoxylin and eosin stain, original magnification ×150.

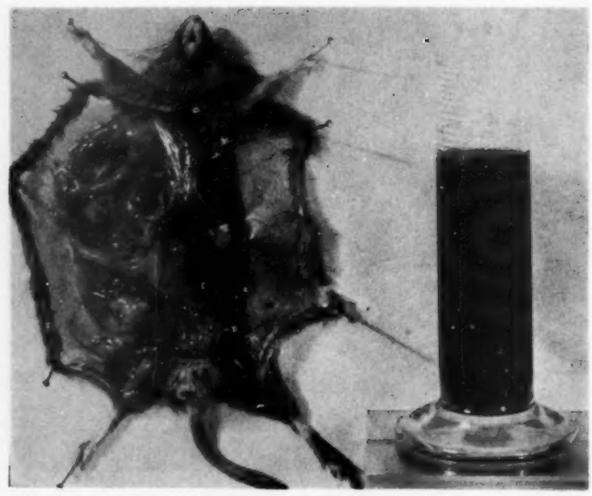


FIG. 16. Large, cystic, subcutaneous tumor transplant derived originally from same "mixed" glioma described in Figure 15. This cystic tumor yielded over 17 cc. of blood-tinged, amber fluid which clotted spontaneously on standing in graduate.

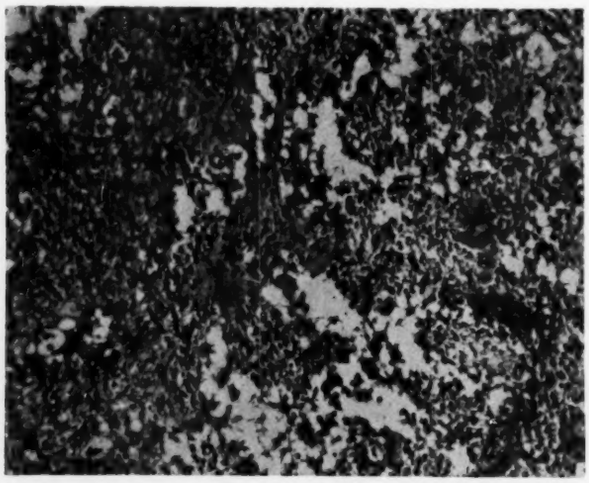


FIG. 17. Photomicrograph of loose fibrillary and cystic astrocytoma of subcutaneous tumor transplant illustrated in Figure 16. Hematoxylin and eosin stain, original magnification ×150.

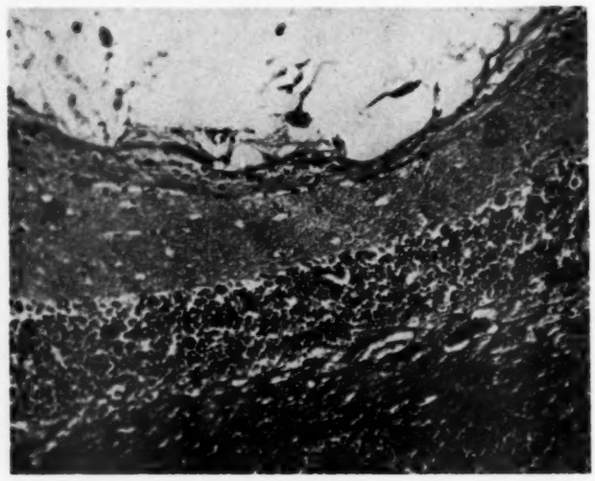


FIG. 18. Photomicrograph of pellet space in cerebellum. Note several giant cells, but no tumor cells at this early stage (less than 200 days after pellet implantation). Hematoxylin and eosin stain, original magnification ×150.

participate. Some of the proliferating cells contain the canary yellow pigment which characterizes the methylcholanthrene used in the experiment. Already there can be noted a wide variation in the appearance of the proliferating glia.

More clearly perhaps can be seen the simultaneous proliferative activity of adjacent glial cells in another animal. (Fig. 20.) Here the yellow pigment of the carcinogen is found in several altered cells which give rise to the tumor. Since the glioma is derived from several different progenitors, all having sprung into proliferative activity almost simultaneously, it is not a "pure" tumor, that is, composed of one cell type, but is rather a mixture of several different cell types. As the tumor continues to grow, its different parts may present rather diversified cellular populations. This depends, of course, on the number and kind of glial elements which have been stimulated to neoplastic activity by the carcinogen, and on the rate of growth of the different glial cells present. It is quite conceivable that the more rapidly proliferating spongioblasts in time may overgrow and replace the more slowly pro-

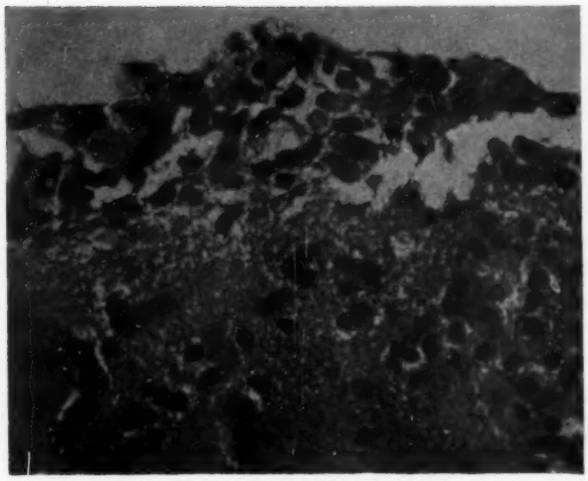


FIG. 19. Photomicrograph of incipient glioma on margin of pellet implanted within lateral ventricle. Several varieties of glial cells are already in the process of proliferation. Hematoxylin and eosin stain, original magnification ×545.

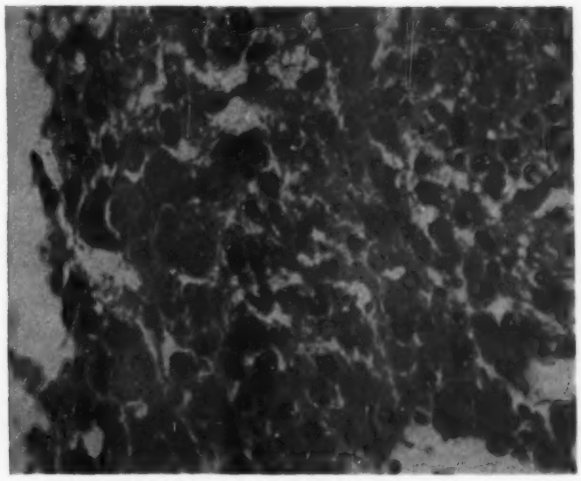


FIG. 20. Photomicrograph of young, carcinogen-induced glioma showing simultaneous proliferation of many different glial cells. Pellet space is on left margin of figure, and adjacent to space there are several large glial cells with coarse granules of carcinogen in their cytoplasm. Hematoxylin and eosin stain, original magnification ×545.

liferating astrocytes. In this view of the histiogenesis of experimental gliomas, the concept of dedifferentiation has a definitely limited role. It can account for the presence of astroblasts or even of spongioblasts in a tumor derived from adult astrocytes, but it need not be invoked to explain the presence of other, totally different gliacytes. The terminology employed to classify a glioma in any stage of its growth depends on the predominance of the glial cell type in the microscopic picture, or on the identification of the most rapidly proliferating cell type present in the tumor.

The Problem of Extracranial Metastasis. It has long been recognized that whereas the more malignant gliomas, such as the medulloblastomas, ependymomas and glioblastomas multiforme, frequently spread along the cerebrospinal axis, they metastasize extracranially extremely rarely. The cerebrospinal fluid may carry implants from deep-seated tumors into the ventricular cavities, or over the convexities of the brain and along the spinal cord. The tumor cells follow the cerebrospinal fluid pathway, lodge singly or in clusters among the trabeculae of the subarachnoid space, proliferate and then invade the neural tissue by piercing the pial membrane or, more rarely, involve the dura by penetrating the arachnoid. Of course, the neural tissue may also be invaded by tumor cells which find their way along the perivascular spaces, these being extensions of the subarachnoid space.

On occasion, also, gliogenous tumors develop in the scalp tissues of patients who have undergone earlier craniotomies, and it is assumed that these subcutaneous new growths are the results of transplanted tumor cells at the time of operation. Likewise, in a very few instances, malignant gliomas have been found in the lungs of patients subjected to operative procedures for brain tumor. In these cases, too, it is plausible that the operations were directly responsible for the extracranial metastases. Here, however, the blood vessels of the brain probably served as the pathway for the spread of the tumor. On the contrary, the unique cases of metastasizing cerebellar tumors reported by Borden and Lewey [8], in which operative procedures were not performed, are probably not examples of gliogenous neoplasms but rather cerebellar sarcomas.

The obvious explanation for the absence of distant metastases from intracranial gliomas is that there is no readily accessible pathway for the spread of these tumors. Aside from the lack of a lymph vascular system in the brain and spinal cord, it has also been noted that glial tumor cells have little or no ability to invade blood vessel walls. This is true even for the most malignant of the gliomas. It is a fact that tumor cells are almost never found invading the vessel walls, and are never seen in the lumens of blood vessels, in the glioblastoma

multiforme, a neoplasm which frequently has ruptured blood vessels and hemorrhage, vascular thromboses, necrotizing vasculitis and vascular proliferation.

That gliogenous neoplasms will grow extracranially with the greatest ease if mechanically transported to such locations has been made abundantly clear in the recital of the subcutaneous transplantation experiments. Years ago, Dr. Freeman and I [9] reported the ease with which the experimental tumors grow by intraocular transplantation in both homologous and heterologous strains of mice. Both by accident and design it has been repeatedly demonstrated that the experimental gliomas grow equally well in the pleural and peritoneal cavities. Within recent years, Dr. James Berkman of our laboratory prepared homogenates of the experimental gliomas in various dilutions and succeeded in producing gliogenous tumor growths in the liver, kidneys, spleen and lungs by intravenous injections into the tails of mice. All these experiments confirm the clinical observations that gliomas will grow extracranially if provided with transportation.

SUMMARY AND CONCLUSIONS

The incidence and radiosensitivity of the various intracranial neoplasms have been briefly indicated.

It was shown that the gliomas constitute the most important group numerically among the intracranial tumors. In addition, they are the most interesting of the tumors because they seem to have sites of predilection within the brain and because they fail to metastasize extracranially. Experimental evidence has been cited which throws some light on these phenomena.

The most challenging problem, however, with regard to the gliomas is the question of their histiogenesis and thus, indirectly, their classification. This problem has been discussed in some detail from the background of pertinent experimental results and can be recapitulated briefly as follows:

Under chemical stimulation many different adult and morphologically distinct glial cells begin to proliferate almost simultaneously to produce a glioma in the animal brain. The resulting tumor is almost never a "pure" glioma composed of cells of one type, but rather a mixture of cells of several different types. In a true sense it is a multipotential neoplasm, that is, it has the cellular composition and the potential to become one or another of the major types into which gliomas are commonly classified.

Transplantation experiments have actually proved the validity of this concept for by judicious selection of explants from a "mixed" glioma, multiple "pure" gliomas may be created by transplantation into homologous animal species. This experience justifies the view that all experimental gliomas are really variants of one tumor type, in the sense that all malignant lymphomas are variants of a single neoplasm.

The principle of multipotentiality is equally applicable to the human gliomas. They, too, like the experimental tumors, are often composed of many different cell types. Some of them, like the glioblastoma multiforme, may even have a multicentric origin in one or both cerebral hemispheres. The human gliomas are also analogous, therefore, to the malignant lymphomas. This being the case, what is the justification for classifying gliomas? The answer is threefold:

1. Clinical experience has shown often that the prognosis in a given case can be made accurately and the proper therapy instituted on the basis of the accepted schemes of classification.

2. In the evolution of a given glioma there usually is developed a characteristic microscopic picture which permits classification. This picture is produced by the predominating cell type, but may be influenced also by vascular proliferation, thrombosis, hemorrhage and necrosis, and sometimes by the appearance of the most malignant of the glial cells participating in the neoplastic process.

3. Attempts at classification are also justifiable on the basis that certain gliomas occur frequently at definite sites of predilection in the brain.

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DISCUSSION

ORVILLE T. BAILEY (Indianapolis, Ind.): It seems to me that there have been two real landmarks in the study of the gliomas: one, the order that Drs. Bailey and Cushing brought out of chaos in their book in 1926, which gave us a set of categories into which it is possible to put most gliomas; and two, Dr. Zimmerman's work, which has extended over a period of years, giving us a new insight into some of the recognized pictures of these separate categories. The findings which Dr. Zimmerman has presented do not suggest that the pathologist faced with a small biopsy specimen of a very large lesion is going to have an easy task. To this one may add the fact that the ordinary laws of repair and tissue reaction also continue and very often, especially at the periphery of these tumors, one may see reactive gliosis, collagenous and other types of repair, all of which must be taken into account in evaluating a biopsy specimen. Dr. Zimmerman has very clearly brought out, and I should like to emphasize, the necessity for making a diagnosis of glioblastoma multiforme in a tumor with many astrocytes if one finds any evidence of dedifferentiation, for if the pathologist does not have the courage to do this, he is not helping the surgeon as he really should.

EDWARD B. SMITH (Indianapolis, Ind.): Dr. Zimmerman has presented some of his own experimental work regarding carcinogenesis or neogenesis produced in the brain by methylcholanthrene and various other coal tar derivatives. I would like to ask Dr. Zimmerman if there are other ways of producing these neoplasms in the same manner in which they have been produced in other parts of the body, for example, by x-ray or any other physical agents? Is there evidence that intracranial tumors have an hereditary factor or any other factor similar to tumors elsewhere in the body?

DR. ZIMMERMAN: It is my conviction that any of these carcinogenetic agents in themselves are really incapable of producing neoplasms. Their use is a stunt which is employed to do something to the nervous system to help create a neoplasm. The reason I say this is because, as you will recall, I mentioned that only in 50 per cent of the most susceptible mouse strains do brain tumors develop. If we take C3H, or dba mice, we can predict that in only 50 per cent of these animals infected with methylcholanthrene will neoplasms of the nervous system develop. If we use mice of the C57 black strain, neoplasms will develop in only approximately 10 per cent of the animals. We have gone so far as to cross breed C57 mice against C3H mice, and neoplasms developed in roughly 25 per cent of the F_1 progeny when injected with the carcinogenic agent. This seems to indicate that there is an hereditary factor. Another important fact is that when the very pellet of methylcholanthrene used in some animal without producing a brain tumor is removed when the animal dies of old age and is implanted into another animal, it may produce the tumor. In other words, it is not the chemical carcinogen as such that produces the tumor, but rather some hidden factor. I do not like to use the word "virus" to indicate that "something" which is stimulated to activity by chemical carcinogens. I do not know that it is necessarily a living agent, but something besides the chemical carcinogen has to be present to produce these neoplasms. We have used radiation on mice and have tried various other methods of producing gliomas, and except for the chemical carcinogens we have not succeeded. We have even found in experiments in which radiation was used in graded dosages on some of these neoplasms that we cannot destroy them once they have been produced with carcinogen. As much as 5,000 roentgen units delivered in a single dose to a glioblastoma multiforme, if it does not kill the animal, will produce a temporary slowing of the growth of the glioma, but growth starts up again at the end of twenty-four to thirty days.

PERCIVAL BAILEY (Chicago, Ill.): I enjoyed this experimental study of a subject with which I was once very much concerned. There is, in addition to the hereditary factor, also some sort of constitutional factors that we do not understand very well. When people first began to produce these tumors in various parts of the body with carcinogenic agents, Dr. William Sweet and I tried to produce gliomas by the use of methylcholanthrene, and unfortunately we chose rats and dogs. In the rats tumors did not develop with one exception, perhaps because we killed them too soon (at six months). In all of the dogs sarcomas developed. In this way we did not produce any gliomas.

I would like to comment on Dr. Zimmerman's tables. I was very interested to see that he showed a percentage of only about 3.9 for unclassifiable gliomas, whereas in Dr. Cushing's series of cases, most of which I diagnosed, there were about 17.9 per cent unclassified gliomas. Of course, that may

be partly due to Dr. Zimmerman's better acumen, but I remember that there was a German pathologist named Scherer who said that there were about 39 per cent that were unclassifiable. It simply shows that one has to use the same characteristics or the same criteria for making the diagnosis of "unclassifiable." That is the general difficulty pathologists have. The clinician always knows whether or not he made the correct diagnosis because the pathologist tells him, but when it comes to the pathologist knowing whether or not he made a correct diagnosis, that is a matter of opinion. We have all had the experience of sending one of these brain tumors to a board of experts of six members and getting five different diagnoses, but that is something medical students should not be told; you learn that as you grow older.

I also noticed from Dr. Zimmerman's tables that the percentage of metastatic tumors in the brain was rather low. Statistics, again, vary a great deal in that regard. In some estimates metastatic tumors run as high as 30 per cent of all intracranial neoplasms, as in the statistics of general hospitals. At any rate, the number of metastatic tumors is going to be much higher in any general clinic than it will in a neurosurgical clinic, as Dr. Zimmerman said, because many of these cases have been screened out before they reach the surgeon. Therefore, actually what percentage is metastatic varies in different statistics. However, I think it is much higher than it was in either Dr. Zimmerman's or Dr. Cushing's statistics, perhaps as much as 15 per cent. If possible, I would like to have Dr. Zimmerman discuss that because he has had more experience in general pathology than I have.

DR. ZIMMERMAN: I disavow any superior diagnostic ability to Dr. Bailey. I have seen many of the tumors in the Cushing collection that have been classified by Drs. Bailey and Eisenhardt as unclassified gliomas. The reason I have a smaller percentage is because I have been freer in giving a malignant classification to many of these neoplasms. In my series, when we have followed the clinical course of patients with unclassifiable gliomas, we found that most of them died within a period of seven to twenty-one months after operation, so that we believed they had at least malignant gliomas. However, regardless of how one classifies them, either as unclassified or malignant gliomas or as I did (by simply calling them glioblastoma multiforme), the one most important point that is brought our by Dr. Bailey's comment is that they are unclassifiable because they are mixed.

I am very glad that Dr. Bailey commented on the number of metastatic tumors. In my New Haven Hospital series we had 13.3 per cent metastatic tumors as against 3.3 per cent in Dr. Cushing's series. This latter series almost exclusively was a surgical series, and at the New Haven Hospital we included, together with our surgical biopsy material, those patients with metastatic brain involvement who came to autopsy from services other than neurosurgery. At Montefiore Hospital where we have about 100 beds for patients with neoplastic disease, many of them with terminal cancer, more than 30 per cent of the tumors in the brain are metastatic. This number, I think, is padded in the opposite direction; our hospital is a catch basin for much inoperable metastatic disease and the patients are kept until they die. At autopsy, therefore, we find many instances of metastases to the brain. I think that is the important difference in the various series. If you deal primarily with a necropsy service, you will have many more metastatic tumors.

Clinical Diagnosis of Supratentorial Tumors*

JOE PENNYBACKER, M.D., *Oxford, England*

From the Department of Neurological Surgery, Radcliffe Infirmary, Oxford, England.

To anyone who has followed the development of neurologic surgery over the past twenty-five years, two features must be evident: the increasing precision of diagnosis and the increased tempo of the work. Many of us can remember the time when intracranial operations were to some extent voyages of exploration and discovery. Today the surgeon usually knows precisely that a tumor is present before he operates, and he often knows its exact location, extent and pathologic nature. He is able to plan his operation accordingly rather than having to improvise as he goes along. This makes for more rapid surgery; and although speed is not an end in itself, a short operation is often better than a long one and, within limits, is better for the patient. As an example, we have found that the risks of wound infection rise sharply after the fourth hour of exposure; and although this is not much of a problem in these days of sulfonamides and antibiotics, even a superficial infection can vastly prolong the period of hospitalization and some patients still die from infection due to resistant organisms.

As each operation takes less time, more can be done in a given period. It is a general experience that each year more patients are seen through the clinic. In our own department, which has been virtually static in size over the past eighteen years, the figures for inpatient admissions has risen steadily from 225 to 410 per annum, and the number of major operations from 125 to something over 300 per annum. As with speed, there is no particular virtue in numbers; however, they may be interpreted as indicating the service which a neurosurgical department provides for the community. We do not have to seek far for an explanation of this increased tempo; better anesthetics, better radiology, sulfonamides and antibiotics all contribute, but more than any other single factor I think cerebral angiography is responsible. Patients with neurologic problems which formerly might have taken days to unravel may now find themselves in the x-ray department within a few hours of admission; an angiogram is taken and the diagnosis made. This is an easy arrangement for the neurologic surgeon in the United Kingdom because in most centers angiography is in the hands of neuroradiologists who soon become highly skilled in technic and interpretation, and thus save a great deal of time and trouble for the surgeon. In some clinics angiography is done as an outpatient procedure. This saves time when the patient is admitted and enables some system of priority for admission to be carried out, a problem which besets most clinics in England.

However, there is one disadvantage which is already becoming manifest, i.e., that we may be relying too much on technics and allowing our senses of clinical diagnosis to atrophy. I think I recognize this in myself and certainly I see it in the young men in training, some of whom are apt to regard time spent in taking a good history and making a full examination as less rewarding than getting the patient off to the x-ray room for an angiogram. In most cases of supratentorial tumors, for instance, this will provide the answer; but when it does not and we are thrown back on our clinical resources, diagnosis may become very difficult. Technics may indeed mislead us, as in the following case:

CASE 1. E. W. (Radcliffe Infirmary, No. 192432), a fifty-one year old woman, was admitted in coma on the night of August 23, 1955.

* Read at the Ninth Annual Symposium on Malignancy given at the Indiana University Medical Center, Indianapolis, Indiana.

She was transferred from another hospital with the history that she had been in good health until the beginning of August when she began to complain of headache and nausea, and her husband noticed that she was a little confused and forgetful at times. She was observed to be sleeping more than usual, and these symptoms led to her admission to the referring hospital. There a right homonymous hemianopsia was revealed but there were no other abnormalities. In particular there was no papilledema, the cerebrospinal fluid was normal as to pressure and content, and x-rays of the skull slowed no abnormality. She gradually became more drowsy and on admission was responding only to painful stimuli. An angiogram taken on the night of admission indicated a tumor in the left parieto-occipital region which accorded with the homonymous hemianopsia. A burr-hole biopsy was performed and one specimen revealed an astrocytoma. In the state that she was in and with an infiltrating tumor in this situation, it was believed that surgery had little to offer and a bad prognosis was given to her relatives.

On the following day, however, she was a good deal better and improved steadily until by August 29th (six days after admission) she was perfectly alert and rational, had no headache and the visual field defect had filled out to leave only a lower quadrant homonymous defect. Another angiogram was taken and there was less displacement than originally, although there was still some shift of the pericallosal vessels across the midline. The sedimentation rate, which had been 105 mm. on admission, had come down to 45 mm. We accordingly revised the diagnosis and suggested as an alternative that this might have been a vascular lesion, e.g., a manifestation of giant cell arteritis. She was allowed to go home on September 5th, free from symptoms, having had no significant treatment.

She was readmitted for review on October 28th (six weeks later); she had had no symptoms during this time and was leading a normal life. There were no abnormalities on examination except the right lower quadrant visual field defect. The erythrocyte sedimentation rate was now 8 mm. Another angiogram was regarded as virtually normal. We believed that this improvement was much more consistent with a vascular lesion than a neoplasm and gave a good prognosis.

She remained well until November 23rd when the headaches recurred and she was readmitted to the hospital. For the first time a slight aphasia was apparent and the hemianopsia was now complete. On this occasion we thought an air encephalogram might be more helpful than an angiogram but she collapsed in the x-ray department and the procedure had to be abandoned. She was then taken to the operating theater where the lesion was found to be an astrocytoma in the parietal and occipital lobes. There was no evidence of recent or old hemorrhage into the tumor.

This case demonstrates how difficult clinical diagnosis can be, but it also shows that too much reliance on a technical procedure can be misleading. If clinical diagnosis is important for the surgeon in the clinic, it is even more important for the general practitioner because it may be all that he has at his disposal, and only by exercising it can he refer his patient to the proper specialist. Most of us have seen the patient who is regarded as having a neurosis or psychosis and has spent some time in a mental hospital before the diagnosis of a brain tumor is made; on the other hand, some of us have had the humiliating experience of operating upon patients who should have undergone psychiatric treatment from the outset. These mistakes will continue to be made, but good clinical diagnosis will certainly render them less frequent.

Tumors of the cerebral hemisphere occur at any age, although they are much less common in children than in adults. Drs. Bucy and Buchanan will also state that in children these tumors are less common than cerebellar tumors, and that whereas many cerebellar tumors are benign almost all cerebral tumors are malignant in the sense that they recur after radical surgery. On the whole they are rather dismal propositions. In adults, on the other hand, a proportion are extrinsic tumors such as meningiomas which can be removed with a reasonable prospect of preservation of life and function. The majority, however, are intrinsic tumors, metastatic cancers or tumors of the glioma group whose special problems Dr. Zimmerman has dealt with so ably.

The meningiomas are discrete, encapsulated tumors, firm to the touch, which grow from the coverings of the brain and compress the brain rather than invade it. They have certain favorite sites of origin, especially where arachnoid villi are present normally, that is to

say they arise especially in relation to the large venous sinuses such as the sagittal and the transverse sinuses.

Next in frequency are those which grow from the meninges in the base of the skull along the olfactory groove, at the tuberculum sellae and along the sphenoidal ridge. They are common, too, on the convexity of the brain without any relation to the large sinuses or dural septums; this is the type which every young neurosurgeon hopes to encounter first. More rarely they occur within the lateral ventricles; for practical purposes these are intracerebral tumors.

Sometimes meningiomas present as a lump on the head, either a huge soft lump or a huge bony one. These large external tumors often have a small intracranial origin and may produce little in the way of symptoms because they decompress themselves as they grow. On the other hand, there may be a relatively small external lump with a massive intracranial growth. Finally, the intracranial tumor may become calcified and its growth either arrested or so slowed down that no action is necessary.

Most of these tumors can be removed with a reasonable prospect of preservation of life and function. They are thus an important group, and I have the impression that their incidence in neurosurgical material is increasing. Dr. Cushing reported an incident of 13 per cent among something over 2,000 verified tumors in 1938, and Olivecrona reported an incidence of 18 per cent among 3,256 verified tumors in 1947. Of the last 100 tumors in our theater, 21 per cent were meningiomas. Olivecrona made the point that meningiomas would be sent for surgery, whereas many of the less favorable types would be weeded out by referring hospitals. Angiography is obviously contributing a good deal in this respect, too.

Apart from meningiomas there are few benign tumors of the cerebral hemispheres. Cholesteatomas occur but are rare, as are colloid cysts. The majority, however, are intrinsic tumors, metastatic cancers or tumors of the glioma group; and although surgery has much less to offer in their management, they all need full investigation to exclude a more favorable type of lesion than the evidence suggests.

Brain tumors begin insidiously, and it may be weeks or months before they cause symptoms sufficient to lead the patient to the doctor. They produce two groups of effects: (1) the general effects of an expanding lesion in a comparatively inexpansible container; and (2) local effects depending on the part of the brain which is involved by the growth. Either effect may be manifest first, or they may appear at the same time and develop *pari passu*.

The general effects can usually be interpreted in terms of increasing pressure within the cranium, although these may be modified in childhood and old age. In children the skull is soft, and the ununited sutures may give before the increasing pressure and allow the skull to expand insidiously and almost without symptoms. There are tables for the normal circumference of the head in children and a tape measure is useful in detecting enlargement which may not be apparent to the naked eye. Tapping the skull may elicit a cracked-pot note from jarring of the sutures. In infants increased pressure may be evidenced both from the size of the skull and from assessing the tension at the anterior fontanel. These simple points are worth mentioning because on many occasions I have seen doctors' children with these signs of increased pressure which have escaped the observation of their own parents. We have recently seen a left frontal glioma, 10 cm. in diameter, in a two and a half year old girl (No. 215253), the daughter of medical parents. She apparently had never had a day's illness until one morning when she awoke complaining of abdominal pain. She was a little listless and vomited once or twice during the day, but was able to get up and play at tea time. Later in the evening she vomited repeatedly and became drowsy. In the early hours of the next morning she went into a state of extensor rigidity and opisthotonos which at first was thought to be a manifestation of tetany, and she was sent to the hospital immediately. She arrived almost moribund, and apart from her general state the only feature which was impressive was the large size of the head: it was 5 cm. greater in circumference than was normal for her age and there was a cracked-pot note on percussion. In retrospect the parents recalled that the child had had two periods of pallor and languor lasting for approximately twenty-four hours at a time within the preceding four months. They had also noticed that she smiled more out of the left side of her mouth than the right, but this was ascribed to a familial facial asymmetry. Both of these features may have been significant.

In elderly subjects the brain is a little shrivelled, and this means that there is a good deal

more room for an expanding lesion to take up before the intracranial pressure begins to rise. Thus a brain tumor can attain a considerable size before any pressure effects are manifest.

A common history is that of a seventy-one year old woman who was admitted because she suffered from drowsiness. She denied any headache; there was no papilledema and the spinal fluid pressure was normal. As she had had two or three falls, her doctor rightly suspected that she might have a chronic subdural hematoma. A parietal burr hole, which was enlarged to allow inspection of the brain, revealed a huge subarachnoid space. A ventriculogram was then taken, and in addition to indicating a tumor in the hypothalamus it also revealed gross shrinkage of the brain.

The most common symptom of increased intracranial pressure is headache, and it is of course especially significant in a person who has not suffered from headache before the present complaint. Slight at the outset, it becomes more severe and frequent, and by the time the patient seeks advice it may be violent. By that time it is usually a throbbing ache, aggravated by anything which further increases the pressure such as venous congestion. For this reason it is often worse on waking in the morning and is aggravated by bending, coughing and straining. When it is severe, the patient can often be seen to be suffering; it differs in this respect from the headache encountered in some neuroses in which the description may be equally dramatic. Also, when it is severe there can be no doubt that something serious is amiss even though the headache is the only symptom and there are no signs. We have just seen a young farmer with a cystic cerebellar astrocytoma. He had been sent to us as an emergency case by one of our recently qualified graduates solely because of the character of his headache. There was no papilledema or abnormalities of any kind on examination.

There is nothing absolutely characteristic of the headache of increased pressure: in many cases, for instance, I have found it impossible to distinguish this headache from that of malignant hypertension. The sphygmomanometer is useful, of course, but it may be that a brain tumor and arterial hypertension coexist. The ophthalmoscope may help, too, but it is not always easy to distinguish between the retinal changes of arterial hypertension and increased intracranial pressure. It is also a fact that a brain tumor may run its whole course without the patient ever having a headache: there may be papilledema and other evidence of increased pressure or there may not, and we are left with supposing that some persons just do not have headache. There is much that we do not know about headache—among other things the explanation of the curious fact that a person with a brain tumor often loses his headache when he becomes blind.

The next effect of increased pressure is on the so-called higher functions of the brain. There is a little failure of memory and concentration, judgment is less keen, and there are slight alterations in behavior and temperament. The patient may be seen at an early stage when he is aware of and has insight into these defects, or we may have to rely on relatives and friends for the assessment. If unrelieved, the disintegration of personality goes on to apathy, incontinence, stupor and coma. These gross abnormalities are late signs and the diagnosis is usually apparent by this time; but if there are no gross focal abnormalities such as hemiplegia, it is remarkable how often they are ascribed to some systemic disorder such as renal failure or cerebral arteriosclerosis.

Although these mental symptoms may occur with tumors in any part of the cerebral hemispheres, they are especially common in the frontal lobe and particularly when both frontal lobes are involved together with the corpus callosum. Tumors in this situation may produce profound dementia before there are any symptoms or signs of increased pressure.

A third group of symptoms of increased pressure relates to the eyesight. One or both abducens nerves may be stretched or compressed to produce a squint and diplopia, and this may be the first objective abnormality. Finally there is the frequent story of attacks of blurring or mistiness of vision, lasting a few seconds at a time. These transient amblyopic attacks are symptoms of papilledema and are of value in indicating how long the patient has had papilledema. They are also a warning of impending visual failure from secondary optic atrophy.

The focal signs and symptoms of a cerebral tumor are often more eloquent because their onset may be more dramatic. However, this is not always true; occasionally we find a person with a meningioma in the anterior fossa who has headache and papilledema but no focal signs except loss of the sense of smell, of which he is not aware until his attention is called to it.

Much more impressive, of course, is the advent of epilepsy in one form or another. Fits occurring in a person not known to be suffering from constitutional or idiopathic epilepsy (i.e., in general, epilepsy manifesting itself for the first time in adult life) demand an explanation. This is a vast subject but perhaps I need only say that there is no clinical difference between the epilepsy due to a cerebral tumor and that due to other causes. However, cerebral tumors are a very common cause of epilepsy arising in adult life, and this possibility must always be borne in mind.

Epilepsy may long preclude any other symptom or sign in a brain tumor. We have recently had in the hospital a woman of sixty-seven whom I first saw seventeen years ago. Her only complaint then, at the age of fifty, was occasional attacks of focal epilepsy involving the right side of her face, during which her speech was profoundly deranged. Each attack lasted for a minute or so, and recovery was prompt and complete. These attacks were described as only an interesting inconvenience because she had no more than three or four a year. She was put on anticonvulsants and the attacks lessened in frequency. She had intervals of three or four years without any attacks at all. She continued to lead a perfectly normal life until approximately four months before the present admission, when she began to be a little aphasic. This progressed steadily to the point where she had difficulty in carrying on a conversation, dealing with her shopping and the like. This complaint brought her into hospital where it was found that she also had very slight right hemiparesis. X-rays of the skull now revealed some calcification in the posterolateral part of the left frontal lobe. There proved to be a solid glioma in this region with a cyst containing approximately 80 cc. of yellow fluid above and in relation to it.

Although the pattern of the epileptic attack in this case was so definite and recognizable to most as indicating a lesion in the lower part of the rolandic area in the speech-dominant hemisphere, this is not always so. In many cases the attack is a generalized convulsion, without warning and without sequelae. It may then be wondered how such an attack qualifies as a "focal sign," and it is "focal" only in indicating abnormal function in one or other of the cerebral hemispheres as opposed to the brain stem or cerebellum. Such attacks arising "out of the blue" in adult patients may be very demoralizing not only for the patient but also for his relatives, and it is often the case that this is the indication for further investigation and treatment. We have recently had a sixty-five year old woman in the hospital who had always been in good health despite moderate arterial hypertension until one day when she had a succession of generalized epileptic attacks. They were eventually controlled with paraldehyde and she recovered completely. Her husband, who had witnessed the attacks, was even more frightened of a recurrence than she was, and her physician referred her for investigation ten days later. At this time she felt perfectly well and there were no neurologic abnormalities of any kind. The arteriogram indicated a large tumor in the right frontal region and at operation it proved to be a meningioma approximately 6 cm. in diameter. This is a common story in neurologic practice, and I tell it to stress again the importance of investigation in cases of epilepsy of late onset. It would have been easy to ascribe the attacks in this case to cerebrovascular disease, and it is a frequent experience that a primary diagnosis is apt to stick. I have seen such cases in which the next development, headache, is ascribed to hypertension; then when intellect and personality begin to suffer, this is explained on the basis of cerebral softening; hemiplegia is ascribed to thrombosis and the final coma to hypertensive renal failure, the real cause of the trouble not being suspected until autopsy records a brain tumor.

The objective evidence of a cerebral tumor may or may not be eloquent. Thus frank hemiplegia or paralysis of an arm or leg needs no looking for, and there is the supporting evidence from the brisk reflexes and the plantar responses. However, defects in sensation are not so obvious. A patient with a tumor of the parietal lobe will usually be able to feel a light touch and painful stimuli, and may insist that sensation is not disturbed. In these cases we have to apply more delicate tests involving discrimination and qualities of differences. These are embodied in the tests for stereognosis, two-point discrimination, the recognition of weights and textures, identification of passive movements and the like. In either case we need to know how these defects have developed: if insidiously and gradually progressive, neoplasm is the likely cause; if suddenly, a vascular lesion is more likely than a tumor. Sometimes, however, slight defects due to a tumor can become worse suddenly, i.e., a slight hemiparesis may

rapidly become a complete hemiplegia. In some cases this is due to hemorrhage into the tumor, but probably not as commonly as we think because often when we operate in such cases we find no evidence of hemorrhage into the tumor. This sudden aggravation of signs may be due to impaction of the brain in, say, the tentorial opening or the foramen magnum, with obstruction to the circulation of the cerebrospinal fluid and probably of the circulation of the blood as well. Whatever the mechanism of these sudden changes, it is generally true that neurologic deficits begin insidiously, are slight at the onset and progress steadily. It is for this reason that a neurologic examination is essential as early as possible so that we have a base line on which to assess later developments.

Although many neurologic defects are eloquent and cannot be missed, there are two which very commonly escape detection. Aphasia is one, and it may be because it is very slight or very gross. Slight degrees of disturbance of the language function may be evident only by the occasional use of wrong or unusual words. The patient may circumlocute or "talk around" a word which he cannot get out. He may be aware of this or his associates may notice that he is not as fluent in conversation as formerly. At the other extreme, the disturbance of the language function may be so gross that the patient speaks jargon; the family may regard it as "nonsense" or "rubbish," and not infrequently this is taken as evidence of dementia leading the patient to a mental hospital. In between these extremes there are moderate degrees of aphasia which may be very obvious in conversation and which can always be demonstrated by simple tests for the four aspects of language function, i.e., the ability to express oneself by the spoken word and in writing, and the ability to understand the spoken and written word. Aphasia is a highly significant symptom because it indicates a lesion of the speech-dominant cerebral hemispheres, and in this connection we must know whether the patient is right- or left-handed.

The other sign which frequently escapes detection is a defect in the field of vision, such as hemianopsia. This is easy to understand because many patients with hemianopsia are not at all aware of it, especially when it comes on gradually. Although slight defects may require accurate perimetry, the gross defects can be detected by simple confrontation tests, and you will be able to say whether the hemianopsia is complete or is in the upper or lower quadrant. If the hemianopsia is incomplete, you can tell by repeated examination whether it is extending or diminishing, and this may be valuable information in diagnosis. These visual field defects are of great importance in the localization of the lesion and they may produce the only objective evidence of localization.

SUMMARY

Some of the common symptoms and signs which cerebral tumors may be expected to produce have been mentioned. It is almost as important to realize that in many patients some of these symptoms and signs may not be present; there may be no headache, no papilledema, no mental symptoms and no gross neurologic abnormalities. The presence of any one of them, however, is sufficient indication to refer the patient to a specialist. I have spoken only about the clinical manifestations; and although these may be adequate for the diagnosis of an intracranial tumor, the surgeon will need more precise information as to location, extent and pathology. In most cases this will mean special investigation such as angiography or ventriculography.

DISCUSSION

CHARLES CURE (Indianapolis, Ind.): One point that Mr. Pennybacker made was of particular interest to me. This was in regard to the woman who survived seventeen years with a glioma in her speech area. One wonders if radical surgery is always warranted after the presence of an intracranial tumor is established. Especially if the major symptom is infrequent convulsions, we stand a very slim chance of curing the convulsions by removing the tumor, and risk the production of a much more severe neurologic deficit. Anticonvulsant medication can frequently alleviate this symptom. Awareness of the presence of a neoplasm is important, but we will occasionally increase the patient's problem by acting too quickly.

On the other hand, early diagnosis is extremely important because the most common indication of an intracranial neoplasm is an increasing neurologic deficit. In order to benefit the patient, surgery must be carried out before the neurologic deficit becomes disabling. It gives the surgeon very little gratification to remove successfully an intracranial tumor in a patient blind from optic atrophy because papilledema due to increased intracranial pressure was not discovered in time.

Infratentorial Intracranial Tumors in Adults*

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THE majority of tumors which develop in the cerebellar fossa occur in children. Inasmuch as these are being discussed by Dr. Buchanan, my presentation will be restricted to those tumors of the posterior fossa which occur in adults. The tumors which develop most commonly in this location after the second decade of life are (1) acoustic neurinomas, (2) meningiomas, (3) hemangioblastomas of the cerebellum and (4) metastatic carcinomas. Each of these tumors presents its own peculiar diagnostic and therapeutic problems and these will be considered.

There are, of course, other tumors which may occur in the cerebellar fossa of adults, but since most of these, such as the epidermoid cysts and paragangliomas, are relatively rare, we shall omit them from the present discussion. Gliomas of the cerebellum, particularly astrocytomas and sarcomas of the cerebellar meninges, also are found in adults, particularly in the third decade of life, but as they differ but little if at all from similar tumors in children (which will be discussed by Dr. Buchanan) we shall not consider them further herein.

Acoustic Neurinomas. These tumors arising from the intracranial portion of the eighth cranial nerve are among the more prevalent brain tumors. They present many difficulties, both diagnostic and therapeutic. One of the most crying needs in the field of neurologic surgery today is for the early recognition of these tumors. These are benign, slowly growing tumors. When their presence is recognized early, before they have become tightly wedged in place between the petrous pyramid, the base of the skull, the cerebellum and the brain stem, they can be removed with little trauma to neighboring structures and often with preservation of the facial nerve. However, when treatment is delayed until the tumor has become deeply embedded in the side of the pons and the various cranial nerves have been compressed and displaced, its removal becomes one of the most difficult and most hazardous procedures in neurologic surgery. Fortunately these tumors manifest their presence early by dramatic symptoms—progressive unilateral deafness and ringing in the affected ear. Usually these are the only symptoms for many years. Only when the tumor has achieved a considerable size does it press upon neighboring structures with sufficient intensity to provoke other manifestations. These consist of (1) paresthesias or pain on the same side of the face from compression of the sensory root of the trigeminal nerve; (2) double vision from compression of the sixth cranial nerve or of the pons; (3) awkwardness with the arm and leg on the side of the tumor from pressure upon the peduncle of the cerebellum; and (4) headaches and vomiting due to obstruction of the fourth ventricle. These are all *late* symptoms indicative of a large tumor and the diagnosis should be made years before this stage is reached. It is surprising that although the vestibular portion of the eighth cranial nerve is almost always involved, dizziness or vertigo is a minor and uncommon symptom with these tumors. In view of the fact that the seventh cranial or facial nerve lies in close proximity to the eighth nerve and leaves the skull with it via the internal auditory meatus, it is also surprising to learn that paralysis of the face is usually absent or very trivial.

The important findings on examination of one of these patients when the tumor is small are a unilateral nerve type deafness and a

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unilateral impairment of vestibular function as revealed by caloric tests. There *may* also be an increase in protein in the cerebrospinal fluid and an abnormal colloidal gold curve. All other findings are late developments and are indicative of the presence of a large tumor. They consist of (1) ipsilateral loss of the corneal reflex, (2) hypesthesia of the face on the same side as the tumor, (3) weakness of the ipsilateral external rectus ocular muscle, (4) ataxia and hypotonia of the ipsilateral arm and leg, (5) nystagmus, (6) unsteadiness of gait, (7) failure to swing the ipsilateral arm when walking, (8) papilledema, and in some cases weakness of the ipsilateral pharyngeal muscles with unilateral loss of the gag reflex and hoarseness with unilateral weakness of movement of the vocal cord. Changes in the roentgenograms of the skull with enlargement of the internal auditory meatus and/or erosion of the petrous pyramid are absent or insignificant in many cases and are usually late developments in those cases in which they are present.

In summary, all patients with a progressive unilateral deafness associated with tinnitus should be carefully examined with the realization that they may be harboring an acoustic neurinoma. If examination reveals a nerve type deafness with impaired vestibular responses on the affected side, such a diagnosis should be seriously considered. If the protein content of the spinal fluid is increased or if any of the other previously mentioned manifestations of such a tumor should be present, the cerebellopontine angle on the affected side should be explored.

The treatment of an acoustic neurinoma is always surgical. Ideally a complete extirpation of the tumor should be made. If the diagnosis is made early this is practically always possible, and in most cases in which the tumor is small the facial nerve can be saved and facial paralysis avoided. Even with large tumors complete extirpation is usually possible but preservation of the facial nerve is much less often possible. The results are undoubtedly much better with total extirpation of the tumor than with the old procedure of merely scooping out the interior of the tumor from within the capsule. However, in older patients with large tumors it is at times best to be content to leave a thin layer of tumor and capsule alongside of the pons. If the extirpation is otherwise thorough in these elderly patients, a recurrence of symptoms from a regrowth of the tumor will usually not occur or not before the passage of many years.

Meningiomas. It is only in recent years that we have realized that meningiomas are by no means strangers in the posterior fossa. These slowly growing tumors which arise from the arachnoid membrane can be divided into three main groups: (1) those lying laterally, (2) those arising from the undersurface of the tentorium nearer the midline, and (3) those arising from the rim of the foramen magnum.

Meningiomas usually grow very slowly over many years. As a result the brain is gradually molded to the gradually increasing mass and few localizing symptoms develop. This is particularly true of meningiomas developing in the posterior fossa. As a result cerebellar meningiomas are among the most difficult of all tumors to diagnose.

Those tumors which lie laterally are particularly apt to be silent. If they lie far laterally there may be no reliable localizing symptoms. Many of these patients will complain of local pain and tenderness over the tumor but localized pain or headache is a notoriously misleading symptom. In most such cases the possibility that a brain tumor is present is recognized only when symptoms of increased intracranial pressure develop—headache, nausea, vomiting and papilledema. The localization is then made by ventriculography.

Other tumors in the lateral part of the posterior fossa which lie somewhat more medially along the petrous pyramid may compress the seventh and eighth cranial nerves, and possibly others as well. Because of the resulting unilateral impairment of hearing, tinnitus and facial weakness, these tumors are more readily recognized. They are often mistaken for acoustic neurinomas. However, the loss of hearing and vestibular function are usually less marked with the meningiomas and the spinal fluid protein is less commonly elevated.

The meningiomas which arise from the undersurface of the tentorium commonly lie nearer the midline and press downward into the cerebellar substance. Again signs of cerebellar dysfunction are usually either absent or mild and the only evidence of the presence of a tumor may be the development of signs of intracranial hypertension. In other instances, however, these tumors push the cerebellar tissue aside as they grow downward and compress the pons, giving rise to signs of multiple cranial nerve palsies and cerebellar

dysfunction. In the absence of signs of increased intracranial pressure, such a picture closely resembles that produced by pontine gliomas in children. One must remember, however, that gliomas of the pons are uncommon in adults and that an adult with such a clinical picture may well harbor a meningioma.

Meningiomas arising from the tentorium not uncommonly invade through that dural structure and produce a mass beneath the occipital lobe, giving rise to a homonymous defect in the visual fields.

Meningiomas of the foramen magnum likewise present a clinical picture which is difficult to recognize. Here the signs are usually those of involvement of the upper spinal cord. As with osseous deformities in this region, such as platybasia, the symptoms most commonly seen are those of spastic quadriparesis and the sensory changes suggestive of a syringomyelia. Usually the intracranial pressure is not increased. Diagnosis here is usually most easily made by myelography.

As with meningiomas elsewhere, ordinary roentgenograms of the skull may reveal a hyperostosis produced by invasion of neighboring bone by the tumor and areas of calcification within the tumor. These are valuable signs when present but they are so often lacking that a negative x-ray of the skull is of no diagnostic value.

As with meningiomas in other locations, the only satisfactory treatment is complete surgical removal of the tumor. In some cases the tumor may be so adherent to structures of great importance that complete removal is impossible or unwarranted. However, in such cases a recurrence is to be anticipated. Fortunately this often does not occur for many years. There is no evidence that radiation therapy is of any value.

Hemangioblastoma. Hemangioblastomas of the cerebellum are benign lesions which usually give rise to symptoms in the fourth decade of life. Most commonly they are large cystic lesions lying within the cerebellum with a small nodule of tumor in one part of the wall. On occasion, however, they are solid, rather vascular lesions which may lie at the posterior angle of the fourth ventricle, in the cerebellar substance or alongside the brain stem.

These tumors are on occasion associated with hemangiomas of the retina and of the spinal cord and with cystic lesions in various intra-abdominal organs. These interesting associations have received so much attention in the literature that the inexperienced are apt to conclude that the demonstration of one or more of them is essential to the diagnosis. Such is not the case. In most instances the cerebellar lesion occurs without the evidence of any other lesions elsewhere.

In those cases in which these tumors are associated with definite manifestations of cerebellar dysfunction or involvement of one or more of the cranial nerves, a localizing diagnosis is not difficult. However, in many cases the patient presents evidence only of a rather rapidly developing increased intracranial pressure. In such cases the proper diagnosis is arrived at only by means of a ventriculogram.

Fortunately these tumors are not only benign but also can usually be removed completely and readily, resulting in a permanent cure. Occasionally, however, the solid, vascular tumors of this group present a very difficult surgical problem and in some instances the surgeon is forced to be content with partial extirpation or only decompression and radiation therapy. The results in such cases are, of course, less satisfactory. The same may be true when these tumors are multiple, either in the cerebellum or in the cerebellum and spinal cord.

Metastatic Carcinoma. Like metastatic malignancies in other parts of the brain, those which involve the cerebellum usually are secondary to bronchogenic carcinomas. In many instances the intracranial metastasis gives rise to symptoms before there are any manifestations of pulmonary disease. It is for this reason that an x-ray examination of the lungs should be a routine procedure in all patients suspected of harboring a brain tumor. However, one encounters from time to time carcinomas of the cerebellum in which the primary lesion in the lung not only gives rise to no symptoms but also is not demonstrated by ordinary x-ray examination of the chest. There are also metastatic carcinomas of the cerebellum which arise elsewhere than in the lung. Most of these will come from the breast but the pancreas, kidney and even the bowel and corpus uteri may be the primary source.

In some instances the surgeon will expose the cerebellar carcinoma before he is aware of its character, and in such cases he will, of course, remove it since the actual removal of a carcinomatous metastasis to the brain is usually a relatively simple procedure. The difficult

decision arises in those cases when one knows the patient with the cerebellar tumor has a primary malignancy in the lung or elsewhere. In the past most physicians have elected not to operate upon such patients and to either let them die while heavily sedated with narcotics or attempt to provide short term relief with radiation therapy. With the passage of the years I have come to believe that such decisions are often a mistake.

Frequently in medicine we cannot provide cures, only palliation. We should not do less for the unfortunate victims of malignant neoplasms. In many cases the one or two comfortable, often valuable years that one can frequently provide by the removal of an intracranial metastatis are greatly enjoyed and appreciated by the patient and by his family. Furthermore, the relief from the intense headache, the misery of nausea and vomiting, and the severe dizziness and uncertainty of gait is in itself more than adequate justification for the surgery. It is my belief that in every case in which one is reasonably sure that the patient has a metastatic intracranial tumor he should operate and attempt to remove the tumor if (1) the patient is in reasonable good physical condition otherwise, (2) there is no clear evidence of multiple intracranial metastases, and (3) there is good reason to believe that the operation will relieve the patient of his distressing symptoms and restore him to reasonably good physical, mental and neurologic health.

Metastatic tumors of the cerebellum are particularly likely to fall in the more favorable category inasmuch as loss of cerebellar tissue at operation is much less likely to result in a permanent or disabling neurologic disability than is loss of neural tissue from many other parts of the brain. In many instances a metastatic intracranial tumor can be removed with very gratifying results and without the patient ever again suffering from a recurrence of symptoms from that source, but dying later from complications elsewhere.

Fortunately, as you have seen, the majority of tumors in the posterior fossa in adults are benign and we are not faced with the harrowing decision discussed previously. The problem in those cases is to recognize the tumor's presence and to recognize it early, at a time when it can still be removed without serious damage to the important nervous structures in the posterior fossa, permanent neurologic disability and blindness.

DISCUSSION

JOHN R. RUSSELL (Indianapolis, Ind.): In view of the great difficulty we have in the diagnosis of tumors of the posterior fossa by clinical means, I wonder if Dr. Bucy has found cerebral angiography to be of any help?

DR. BUCY: I did not touch upon diagnostic neurosurgical methods particularly because I did not think they were of too great interest to this audience. However, I have not found the use of angiography of great value in the diagnosis of posterior fossa lesions, and I have found vertebral angiography on occasion to be a very dangerous method of examination with tumors in that location. Other physicians have apparently been more fortunate than I, and there are several reports in the literature of the diagnosis of acoustic neurinomas and meningiomas with vertebral angiography. Carotid angiography is, of course, of no value in visualizing tumors in the posterior fossa.

JOE PENNYBACKER (Oxford, England): Vertebral angiography on the whole is a bit more tedious than carotid angiography, but I cannot say that we have had the misadventures that Dr. Bucy described. I entirely agree that in many cases it is not at all necessary, but there are a few instances when I believe you can obtain particular help from it—one is with hemangioblastomas, the solid ones which may be a very formidable problem surgically. If you can obtain information from a vertebral angiogram as to where the blood supply arises, it may make a considerable difference in the operation. Let us suppose that a hemangioblastoma is disclosed at primary operation in the lower part of the cerebellar lobe. If reoperation is performed later, you might find that the blood supply is coming from the upper surface of the tumor. We have had several such cases in which angiography made a great deal of difference in our ability to remove the tumor without doing too much damage. I would agree that, as with carotid angiography, these are not technics that can be lightly undertaken. They must be undertaken in consultation with neurologists, neurosurgeons and neuroradiologists. Anyone who has seen many of them will certainly know about cases in which persons with a neurosis or hysteria have been subjected to arteriography and developed the complications that Dr. Bucy described.

E. VERNON HAHN (Indianapolis, Ind.): It is worth while to emphasize the importance of posterior fossa tumors in adults. The frequency with which meningiomas of the posterior fossa cause unimpressive symptoms and signs should be remembered by all of us when we examine patients with vague persistant headaches, since these lesions respond so well to surgical management.

Intracranial Tumors in Infancy and Childhood*

DOUGLAS BUCHANAN, M.D., *Chicago, Illinois*

TWO-THIRDS of the intracranial tumors which give clinical signs before puberty arise below the tentorium. In adults the reverse is true. The pathologic types which tend to occur in set locations within the central nervous system do not differ with age. Meningiomas are rare in childhood and adenomas of the hypophysis are almost unknown. The primary neoplasms which occur in childhood can be localized as follows: below the tentorium: (1) the cerebellar hemispheres, (2) the cerebellar vermis, (3) the cavity of the fourth ventricle, and (4) the pons and medulla; above the tentorium: (5) the optic nerves and chiasm, (6) the hypothalamus, (7) the thalamus, (8) the pineal body and quadrigeminal plate, and (9) the cerebral hemispheres.

DIAGNOSIS

The most common symptom of an intracranial tumor in a child is vomiting. The most common sign is a cracked tone on percussion of the skull. Tumors of the pons and some tumors of the cavity of the fourth ventricle may be associated with vomiting but without evidence of increased intracranial pressure. Since a child's skull can enlarge easily to relieve rising pressure, headache is not a common symptom. The majority of tumors which appear before puberty occur in the posterior fossa and the most common sign of such tumors is unsteadiness in walking. Children who vomit and are unsteady, without the indications of an acute feverish illness or the obvious after-effects of a severe head injury, usually have an intracranial neoplasm below the tentorium. With such a symptom and such a sign, if percussion of the skull gives cracked resonance, the diagnosis of intracranial tumor may be made easily and quickly. The usual triad which is given for the presence of an intracranial tumor is headache, vomiting and papilledema. In children, however, the proper triad is vomiting, unsteadiness and a positive Macewen's sign.

Since most tumors arise in the posterior fossa, convulsions are not common. The association of frequent major convulsions with increased intracranial pressure and ataxia of the trunk is more often evidence of a toxic encephalopathy or of acute meningoencephalitis than of a tumor of the posterior fossa. Even lateralizing or localizing convulsions in a child do not have the connotation of such attacks in adults. There are many children who have their first few convulsions as lateralized attacks and then have full major convulsions with movement of all four limbs. A one-sided attack, whether this is the first seizure or is frequently repeated, is not evidence of an expanding lesion in the opposite hemisphere. It is evidence of an anatomic lesion in the appropriate region, but does not indicate that this lesion is increasing in size. The convulsion which has a true Jacksonian march from the periphery to the center of a limb is usually evidence of an anatomic and expanding lesion in a child as in an adult.

It has long been known, although not well understood, that most children who have intracranial tumors are quiet and well behaved and remarkably mature in all their actions. This is so obvious that physicians and nurses with some experience in this field are constantly aware of it. This is not a change which comes with increase in pressure in the later stages of the illness. It is usually present before any specific signs or symptoms are established.

Accurately to distinguish a primary intracranial tumor from other causes of increased intracranial pressure and defect of local

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function is complicated and difficult but depends on a few general principles. Children are so prone to acquire infections of the middle ear and respiratory tract that the occurrence of such an illness will not distinguish an abscess from a tumor. A child who has an intracranial abscess, however, looks pale and ill while a child with a primary tumor is often remarkably robust.

Lead encephalopathy may produce all the signs of a tumor of the cerebellar vermis with truncal ataxia, papilledema and separation of the sutures of the skull, but major convulsions which are so common with lead poisoning are almost never present with a tumor of the posterior fossa. Basophilic stippling of the red cells of the peripheral blood may be absent with lead intoxication, but roentgenograms almost always reveal deposits of metal at the growing ends of the long bones. Proper interpretation of such lines of increased density is difficult, however, and the only test for lead poisoning which is certain is chemical or spectographic proof of abnormal amounts of lead in the peripheral blood.

The clinical picture of tuberculous meningitis is not often confused with that of a primary tumor but such infection may produce papilledema and separation of the sutures of the skull.

Acute non-septic meningoencephalitis may have for a time unilateral signs of hemiplegia with hemianopsia. The meningeal component usually gives the clue to the diagnosis but some primary tumors do have an associated cellular reaction in the spinal fluid. This is more common, however, with tumors below the tentorium.

Because most tumors are in the posterior fossa, ventriculography and angiography are of less specific value in children than in adults. Both may demonstrate blocking of the aqueduct, but only with the highest technical success is localization possible in the posterior fossa. Since present technics of electroencephalography record electrical discharges from the superficial layers of the cortex, their frequent failure to demonstrate or to localize intracranial tumors in children is not unexpected.

PROGNOSIS

The only type of intracranial tumor in childhood with which complete recovery and freedom from recurrence may be expected is the astrocytoma of the cerebellar hemisphere. Lindau's disease with the vascular tumor in the same region of the cerebellum seldom gives clinical signs before puberty. Other posterior fossa tumors, either from their pathologic structure or their anatomic situation, cannot be removed completely. Meningiomas, astrocytomas or oligodendrogliomas of the cerebral hemispheres are open to possible removal but they are frequently associated with contralateral hemiparesis which is permanent, and with lateralized major convulsions which are most intractable. With the exception of the rare cysts of the third ventricle, other neoplasms above the tentorium cannot be removed with restoration of normal function. Surgical relief and control of advancing intracranial pressure and radiation of the tumor is all that is possible with most of the intracranial tumors of childhood, but this frequently gives great relief and remarkable restoration of function. The prognosis for useful although temporary improvement and for life itself is controlled by at least two obvious variables, anatomic position and pathologic structure.

TUMORS OF THE CEREBELLUM

Tumors of the cerebellum release abnormal neurologic signs which can be separated as the syndrome of the cerebellar vermis and the syndrome of the cerebellar hemisphere.

Tumors of the vermis have as their characteristic pattern gross ataxia of the trunk and legs with little ataxia or incoordination of the hands and arms. The deep reflexes are reduced or absent, the knee jerks may be pendular in type and there is generalized hypotonia. Voluntary movements of the legs reveal both ataxia and incoordination but there is no defect in appreciation of vibration or of change in position of the toes or feet. There may be no nystagmus in voluntary movements of the eyes, but if it is present it is rapid, fine and symmetric.

The syndrome of the cerebellar hemisphere is hypotonia, ataxia and incoordination of the ipsilateral extremities with the defect greater in the arm than in the leg. The deep reflexes are reduced on the affected side but the plantar responses may be extensor. Nystagmus is almost always present and this is slow and coarse to the side of the lesion and fine and rapid to the opposite side. The signs of abnormal cerebellar action are released only in movement, and if the affected limbs are sup-

ported and the child is at rest, no ataxia or incoordination is revealed. The irregular movements of Sydenham's chorea are sometimes confused with cerebellar ataxia, but the jerking of chorea is present even when the limbs are fully supported. Voluntary movement does not suppress the irregularity of chorea but reveals some cerebellar hypotonia in addition. In chorea mollis, however, in which there is almost no jerking at rest, it is remarkable how closely the irregular voluntary movements can simulate a pure cerebellar ataxia.

The tumor which grows in the vermis is usually a medulloblastoma. This has no capsule, grows rapidly and seeds easily throughout the central nervous system. Children affected are most often about three to five years old. The characteristic tumor of the cerebellar hemisphere is an astrocytoma which grows slowly and is encapsulated with a tendency to cyst formation. Signs and symptoms of increased pressure ultimately appear with both types of tumor. These come within a few weeks or months with a medulloblastoma, but are often delayed for some years with astrocytomas.

Astrocytomas may be removed completely. There is then rapid recovery of normal physical control and a normal life expectancy. Medulloblastomas having no capsule cannot be removed completely by operation. Partial removal to decompress the aqueduct and the fourth ventricle is followed by x-ray treatment to the posterior fossa and the spinal axis. This usually produces great improvement but after two to three years the signs appear again. The response to x-ray treatment is then much less and life expectancy is two to four years.

TUMORS OF THE FOURTH VENTRICLE

Most tumors in this region arise from the ependymal lining of the cavity. They may grow downward from the anterior medullary velum, or grow up from the floor of the fourth ventricle often in the region of the obex. Since they grow into a cavity, many will increase in size almost to fill the ventricle before they produce signs or symptoms, but those which grow downward from the upper end of the fourth ventricle may obstruct the lower end of the aqueduct while still quite small.

The most common symptom of such a tumor is vomiting. This is not associated with nausea or abdominal pain and is not related to infection or to injury. Such sudden and unexplained vomiting may occur many times over many months before there are any signs of ataxia, hypotonia or papilledema. The ataxia, the incoordination and the hypotonia tend to be symmetric and in many ways are similar to those defects seen with a tumor of the cerebellar vermis. Although nystagmus is often absent with the syndrome of the vermis, it is almost never absent if the tumor arises in the cavity of the fourth ventricle.

These tumors are not malignant but their anatomic position is such that they cannot be removed completely. Opening of the aqueduct and partial removal of the tumor is all that is possible. Although they grow slowly, radiation has some effect on their development, and for this reason the life expectancy of any child with such a tumor is indefinite.

TUMORS OF THE BRAIN STEM

These are almost always tumors of the glial series which originate in the central regions of the pons and which from their location and from their infiltrating nature remain hopeless problems for surgical treatment. They grow rapidly and soon produce an extensive defect in action of the lower cranial nerves and of the pyramidal and cerebellar systems.

The first sign of such a tumor is often the sudden appearance of strabismus from the loss of outward movement in one eye. This is soon followed by weakness of movement of the face, unsteadiness and difficulty in swallowing. The defective movements of the eye and face become bilateral but asymmetric, the unsteadiness advances to a reeling ataxia, and there is increasing difficulty in speaking and in swallowing. These changes come without fever, without convulsions and without coma or any blunting of understanding. The pons may be enlarged by the tumor to a remarkable size before blocking of the circulation of fluid produces any papilledema, vomiting or headache. Although such signs may follow an acute and specific illness, the preservation of normal understanding in spite of the most devastating defect in mechanical performance is a characteristic and startling feature of such tumors. This alone would exclude the diagnosis of acute encephalitis so often made. It is obvious that the signs are related to the pons, and when a child has a chronic and advancing paralysis of the lower cranial nerves the cause is almost always a primary neoplasm.

These tumors have been classified as astrocytomas, oligodendrogliomas, spongioblastomas and as glioblastomas multiforme. Despite their characteristic cell types, they all grow rapidly and infiltrate widely although they do not spread to other portions of the brain.

They are sensitive to radiation and such treatment may produce a remarkable improvement for many months. Within a year, however, the signs and symptoms appear again and the life expectancy is seldom more than two to three years.

TUMORS INVOLVING THE HYPOTHALAMUS

Adenomas of the hypophysis, meningiomas of the dorsum sellae or of the sphenoidal ridge, chordomas and epidermoids so seldom give signs or symptoms in childhood that they can be omitted from discussion. Craniopharyngiomas and gliomas of the optic chiasm are the tumors of this region which are found in children.

Craniopharyngiomas arise from remnants of Rathke's pouch and they are epithelial in character. They may grow to a remarkable size and often outgrow their blood supply, producing a central necrosis with formation of cysts. They press downward on the optic chiasm and upward on the hypothalamus.

They usually give signs of their presence by the age of six or seven years, although they may remain clinically silent until middle life, or grow so rapidly that they are detected before the age of two. In the youngest group, the clinical introduction is by failing vision with attacks of vomiting. Older children have episodes of headache with vomiting before there is much loss of vision. Adults usually have as the first signs defects in vision and changes in libido and the menstrual cycle.

Affected children are small and frail when young, but obesity gradually develops and gives for a short time a false appearance of improving strength. There are no episodes of erratic behavior and intellectual ability is not changed. Indeed, affected children are usually cooperative and well behaved, and have a strange air of maturity in their speech and actions.

Somnolence is a common symptom difficult to appreciate as an abnormality until it reaches the depth of hibernation. Since the visual loss is the result of pressure on the chiasm and on the optic nerves, any variety of defect in the visual fields can follow. For the same reason there may be a combination of primary optic atrophy and papilledema. These tumors increase the size of the sella turcica and erode the posterior clinoids. They almost always have small areas of calcification in their walls. Roentgenograms reveal these defects and when all are present the diagnosis can be made with certainty. Calcification is very rare in adenomas of the hypophysis and such tumors are almost unknown in children.

Although these tumors have a capsule, their position between the carotid arteries prevents their complete surgical removal. The operation of choice is Torkildsen's maneuver to by-pass or prevent any blocking of the cerebrospinal fluid circulation.

Radiation to the chiasm or hypothalamus will usually improve central and peripheral vision.

Thyroid has little action but cortisone in doses of 20 to 60 mg. each day will relieve much of the metabolic defect. It can be given continuously for many months with much advantage, but should be stopped if there is evidence of infection.

The older the child when signs of increased pressure appear the longer is the life expectancy, but only a few live more than five years when such signs appear before the age of ten. Fröhlich's famous patient, however, was twelve years old when the diagnosis was made and twenty years old at the time of partial removal of the tumor through the nasal route. In 1940 Fröhlich reported to a meeting that he believed the patient was still alive.

TUMORS OF THE OPTIC NERVES AND CHIASM

These may be solitary tumors or may be part of generalized neurofibromatosis or of tuberous sclerosis.

The most common signs which these tumors produce are primary optic atrophy with defective central and peripheral vision. These may be present for months or years before any signs of increased intracranial pressure appear. When primary optic atrophy develops in a small child without the retinal and macular changes of cerebromacular degeneration, the cause is almost always a neoplasm of the optic chiasm. When the skin changes of neurofibromatosis or of tuberous sclerosis are also present, the diagnosis is almost certain.

Since the overgrowth of bone that is found in Albers-Schönberg disease is present *in utero*, the associated optic atrophy and defective vision are present at birth. Tumors of the chiasm, however, do not usually produce visual loss before the age of two years. Chiasmal tumors produce hypothalamic signs of obesity, defective growth, somnolence and hypotension which are of the same quality as those associated with craniopharyngiomas. Both of these types of tumor can usually be diagnosed with certainty from appropriate roentgenograms. With both, there is increase in size of the sella turcica and erosion of the clinoids. Gliomas of the optic nerves and chiasm produce enlargement of the optic foramens, and craniopharyngiomas have calcifications above the sella.

Tumors of the chiasm are not amenable to surgical treatment since any attempt at removal would produce complete blindness. X-ray therapy to the chiasm is given although decompression or some form of by-pass operation may be needed as a general measure. These tumors are of the glial series and are not neurofibromas, but unlike the spongioblastomas of the pons they grow slowly and change little for many years.

TUMORS OF THE THALAMUS

Tumors of the thalamus are not common, but they occur more often in children than in adults. Apart from the general signs of increased intracranial pressure their local signs are few. Although changes in perception of cutaneous sensation might be expected on theoretic grounds, such changes are almost never demonstrated. The most characteristic sign is a contralateral ataxia with some of the quality of Sydenham's chorea, but the irregular movement is more rapid and finer in range and can more accurately be compared to the movements of electric chorea. The affected limbs are weak, there is mild spasticity with increase in the deep reflexes, and an extensor plantar response. In this spasticity is the difference from the weakness of chorea, for there the reflexes are reduced and the picture is of hypotonia or flaccidity. These tumors grow slowly and are usually astrocytic in their type. They may be bilateral and remarkably symmetric, and like chiasmal tumors may be part of the general abnormalities of phakomatoses. As would be expected from their location, hypothalamic signs may appear. These come late, however, and somnolence is usually more marked than obesity.

Surgical removal of these tumors is difficult and the results so poor that x-ray therapy is used with some form of decompression.

TUMORS OF THE PINEAL BODY

Although tumors in this region may occur at any age they are more frequently found in children. Their pathologic types are varied but the most common are teratomas, pinealomas, pineoblastomas and gliomas. They press down on the quadrigeminal plate, forward into the posterior portion of the third ventricle and backward against the cerebellum. While still quite small, they may occlude the aqueduct of Sylvius and produce increased intracranial pressure.

They have specific signs of which the most dramatic is precocious puberty with general physical overgrowth. Since there is no evidence that the pineal has any hormone production, this is presumably the result of hypothalamic overaction. Pressure on the superior colliculi results in absence of upward movement of the eyes and unequal dilatation of the pupils with loss of the pupillary reaction to light. Pressure on the inferior colliculi produces a fluctuating defect in hearing which is both central and peripheral in type. The other neurologic signs are those of any midline tumor, papilledema, bilateral pyramidal signs and ataxia of the trunk and legs. Pineal teratomas frequently contain sufficient calcium even in childhood that they can be recognized by roentgenograms. Calcification of the pineal from any cause is rare in children. It is found most often in neurofibromatosis, but usually with calcification of the choroid plexus in addition. Isolated pineal calcification in a child is usually evidence of a pineal tumor.

These tumors are so situated that surgical removal is most difficult. Major vessels such as the great vein of Galen must be sacrificed for their exposure and hemiparesis and hemianopsia usually result. Increased pressure is controlled by Torkildsen's maneuver, and although most tumors of the pineal are only slightly sensitive to radiation, this is given in the absence of any more effective treatment.

TUMORS OF THE CEREBRAL HEMISPHERES

Primary tumors of the hemispheres occur in children and may be of the same pathologic

types as those seen in adults. They so seldom give signs before the age of two to three years, however, that the diagnosis of primary intracranial tumor in an infant who has a one-sided weakness or a history of one-sided convulsions is usually incorrect. In the second half of childhood their presence may be suspected even in the absence of signs of increased pressure if there are local convulsions with a Jacksonian march. Meningiomas seldom produce hyperostosis of the skull in children but they are sometimes associated with a defect in both tables which is remarkably similar to that of Hand-Schüller-Christian disease. Oligodendrogliomas of the hemispheres may be calcified and may throw shadows on roentgenograms, as in adults. The shadows are usually very faint and fine, and stereoscopic photographs are necessary for their accurate localization within the substance of the hemisphere.

The limitations which are inherent in the neurologic examination of a child are obvious when an attempt is made to localize a lesion of the cerebral hemispheres. It is seldom possible accurately to examine the fields of peripheral vision, to demonstrate aphasia except of the crudest type or to evaluate the perception of the finer modes of sensation. If convulsions occur, they seldom have any Jacksonian march or are even lateralized. Forced grasping, euphoria and confabulation, which may be the key to localization in an adult, are almost never present in a child. Specific signs are usually limited to some asymmetry of the deep reflexes or to the presence of a fleeting extensor plantar response. Most often tumors of the frontal lobe are diagnosed by the absence of any fixed and local signs. Tumors of the parietal and temporal lobes are usually accompanied by a slowly advancing, contralateral hemiparesis with periodic convulsions which may be generalized or lateralized. If the convulsions have a Jacksonian march, the localization can usually be made to the region of the central fissure. In a tumor of the temporal lobe, contralateral facial weakness of the cortical type usually precedes weakness of the appropriate arm or leg, and gross defect of the visual fields can usually be demonstrated. The localization of a neoplasm to the occipital lobe is difficult since fine subjective and objective defects in vision which are so valuable in localization can almost never be demonstrated with accuracy. When such a tumor has grown sufficiently to invade the adjacent parietal cortex, the appropriate signs of a contralateral hemiparesis appear.

The treatment of tumors of the hemisphere depends on localization, lateralization and pathologic type. The decision for surgical removal or palliative treatment by radiation after partial removal and the relief of pressure rests on the same principles with children as with adults.

DISCUSSION

DWAIN N. WALCHER (Indianapolis, Ind.): We have made the diagnosis of cerebellar encephalitis in a series of patients. There is infrequent reference to this condition in the literature under the terms acute cerebellar ataxia and cerebellar encephalitis. If Dr. Buchanan accepts this as an entity, I would like to hear him discuss his own observations, and the difference between it and cerebellar tumors in children.

DR. BUCHANAN: The acute illness sometimes described as cerebellar encephalitis has spectacular signs and a characteristic history. It occurs in young children without any preceding acute illness or accident. The first sign is unsteadiness in walking. This increases and within two or three days the child can no longer walk alone. He lies in bed and resists all attempts made to move him. He lies face down, holding the bedclothes with both hands. If pulled forcibly from this position he will turn at once to face the adult and hold to the adult's clothes as if he had severe unsteadiness and vertigo. This acute phase lasts about two weeks. After the first few days a characteristic movement of the eyes appears. The eyes dance as if suspended on hairsprings. This movement is rapid and spectacular. It is not a nystagmus. There is no papilledema or optic neuritis. The deep reflexes are reduced and there is a general hypotonia. Despite the severity of the unsteadiness and its relatively sudden onset, there is usually no fever. The meningeal component of the illness is mild. The cerebrospinal fluid may contain three to twenty cells or may be normal in every respect. Gradually the child recovers the ability to stand and then to walk. The illness usually lasts from three to six months. The pathogenesis is not yet known. It may be related to a virus infection but no specific type has yet been recognized. This illness of young children is so spectacular in its signs of severe vertigo and dancing movements of the eyes that it can be distinguished easily from the effects of a primary tumor in the posterior fossa.

Reliability and Limitations of X-ray Diagnosis of Intracranial Neoplasms*

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THE use of roentgen procedures in the diagnosis and localization of brain tumors is ever increasing and yet the incidence of positive findings is decreasing. This apparent paradox is due to the fact that patients are being studied much earlier in the course of their disease before well developed classic x-ray signs are present. This means that the x-ray studies must be carried out and interpreted with greater and greater skill.

All patients with a suspected brain tumor should have plain films of the skull before other more complicated procedures are undertaken. This initial study should be quite extensive, usually including six views as a minimum. Of those patients having brain tumors of all types, about 40 per cent will have some abnormality on these films and about 20 per cent will have localizing findings. Thus, in the majority of the cases, further x-ray procedures will be necessary, meaning either pneumography or angiography. Sometimes these studies are utilized even when the plain films are of definite localizing value in order to obtain more detailed information about the tumor preoperatively. Because these technics are not without risk, they should be performed by well qualified neurologists, neurosurgeons or radiologists and only when real indications are present which outweigh the dangers. The value of these procedures is directly proportional to the exactness of the technics and the astuteness of the interpreter. In the best clinics the addition of these special procedures to the routine plain films will result in a correct localization in about 90 to 95 per cent of all brain tumors.

PLAIN FILMS

The roentgen findings of brain tumors can be divided into three broad categories:

1. Roentgen evidence of increased intracranial pressure.

In the adult this is evidenced primarily by decalcification, which is most easily detected in the region of the sella. This decalcification is most readily detectable in the dorsum sellae but may also take place in the floor of the sella and can be recognized in the sphenoid wings and orbital roofs on rare occasions. The length of time required for this decalcification to develop is extremely difficult to ascertain inasmuch as the exact onset of the tumor cannot be determined. Very likely a period of two to three months is necessary in the usual type of brain tumor in which the onset of increased pressure is not acute and the intensity of the pressure is not great. A fair degree of experience is necessary to interpret correctly the changes in the sella due to increased pressure. The greatest error takes place in the direction of over-reading. This is especially true in older people in whom there may be slight generalized decalcification of all of the bones, and it becomes particularly striking if the skull is rotated slightly at the time the film is made so that the dorsum sellae is seen in a slightly oblique rather than a true lateral projection. In these circumstances the appearance of increased pressure is closely simulated when the pressure is actually not increased.

In children the first signs of increased pressure are a spreading or separation of the sutures which is usually most easily recognized first in the coronal suture, then in the sagittal suture and last in the lambdoid suture. This spreading of the sutures of the cranial vault does not occur after about the age of fifteen or sixteen years. Decalcification of the sella may also occur in children but usually develops some time later than the separation of the sutures.

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Increased digital markings as a sign of increased pressure is often unreliable inasmuch as the digital markings in children are normally very prominent. Consequently, increased or prominent digital markings in the absence of separation of the sutures or decalcification of the dorsum is not an accurate sign of increased pressure.

In children who have chronically increased pressure of a low grade, it is possible to produce an enlargement of the sella which simulates an intrasellar tumor. Also in these cases there may be increased digital markings throughout the skull, usually without significant or recognizable separation of the sutures. In order to obtain these findings, it is essential that the pressure be of a low grade and exist for a period of years.

It is occasionally possible to have x-ray findings of increased pressure without clinical evidence of choked discs. Much more commonly, however, the x-ray signs of increased pressure are absent and the clinical signs of increased pressure are present.

2. Displacement of normal structures.

The pineal is calcified in about 60 per cent of adults. In many instances of brain tumor, hemorrhage or abscess, the calcified pineal will be displaced away from the lesion. A shift from one side to the other is the most easily recognizable type of displacement and is the most reliable. There is a normal variation in the position of the pineal with reference to the midline of approximately 3 mm. to either side. A shift of more than 3 mm. most often indicates a space-taking lesion on the opposite side. A shift of the pineal in the anteroposterior or vertical direction is more difficult to determine and the significance is less accurate.

The falx may be calcified as well as the chorioid plexus on the two sides, and very occasionally a displacement of these structures may be of diagnostic significance. A displacement of any of these normally calcifying structures is admittedly of only gross localizing value but may be of considerable significance in influencing the decision as to whether or not further more complicated procedures should be done.

3. Localizing findings.

There are some localizing findings on plain films which are very accurate. These are (1) calcification in the tumor, (2) bone production, (3) bone destruction and (4) increased vascularity in the skull.

Calcification is reported to occur in about 15 to 20 per cent of all tumors. Very likely this high incidence which was recorded several years ago has now been reduced to 10 to 15 per cent. The calcification can occur either in the form of large, dense masses or in small, discrete, punctate collections or in linear streaks. Some tumors calcify more often than others. For example, craniopharyngiomas have an incidence of calcification of about 60 to 70 per cent and oligodendrogliomas calcify in about 50 per cent, whereas calcification in a glioblastoma multiforme is quite rare. Although some success might be obtained in estimating the type of tumor from the appearance of the calcification, this is usually not a pathognomonic sign. In some instances the type of calcification plus its location may give fairly good evidence of the type of tumor, such as stippled suprasellar or curvilinear calcification in a craniopharyngioma. Lipomas of the corpus callosum frequently present a rather characteristic ring-like type of calcification surrounding an area of lessened density.

Bone production arising from the inner table of the skull or the base of the skull occurs fairly often in association with a meningioma. This is the only type of brain tumor which can stimulate bone production. A rather localized osteoma may be found at the base of a soft tissue tumor or there may be a rather diffuse increased density and thickening of bone over a relatively large area frequently involving the wings of the sphenoid. These bone changes, when characteristic, are quite pathognomonic for the localization of a meningioma.

Bone destruction can be due to direct pressure erosion or can occur secondary to invasion of the bone by the tumor. Direct invasion occurs only with the meningiomas, but the extent of destruction is usually not marked. Pressure erosion of bone occurs with an occasional intracerebral glioma, quite often with brain cysts and is, of course, the common finding for pituitary adenomas and a fairly frequent but not as common a finding in acoustic tumors. Perhaps 70 to 90 per cent of pituitary tumors show characteristic changes in the sella. This consists of an increase in the diameters of the sella with a depression of the floor into the sphenoid sinus, thinning and posterior tilting of the dorsum sellae, and

occasionally a thinning or elevation of the anterior clinoids. Acoustic tumors may cause enlargement of the internal auditory canal, but frequently a diffuse decalcification or destruction of the medial end of the petrous pyramid takes place and the acoustic neuroma cannot be differentiated from some of the cerebellopontine angle meningiomas. Perhaps 50 per cent of acoustic tumors show suggestive or characteristic findings on the plain films.

Increased vascularity of the bones of the calvarium is occasionally found in association with meningiomas. This is a type of change which is not easy to recognize since there is such a wide variation in the normal vascularity of the skull. A diagnostically reliable increased vascularity probably does not occur very often and one should be very cautious in interpreting vascular markings as being abnormal.

PNEUMOGRAPHY

The injection of gases into the ventricular system either by direct ventricular puncture through the brain (ventriculography) or by indirect filling through lumbar puncture (encephalography) usually results in very accurate localization of most intracranial space-taking lesions. There are definite indications and contraindications for the use of these procedures which are not within the scope of the present paper. The many roentgen technics to be employed in order to obtain maximum information must also be deleted. In general, there are four ways in which lesions can be localized following injection of gases.

The most common localizing abnormality is a displacement and deformity of the ventricular system. A mass in one cerebral hemisphere will displace the ventricles toward the opposite side and will usually deform the ventricle on the side of the tumor in a characteristic fashion which will quite accurately localize the mass. Small superficial masses and diffuse masses, such as a subdural hematoma or an infiltrating astrocytoma, cause the greatest difficulty in localization.

The second and perhaps the next most common type of abnormality is an obstruction of the flow of ventricular fluid resulting in a uniform enlargement of the lateral ventricles. A lesion in the third ventricle, in the region of the aqueduct, in the fourth ventricle, in the basilar cisterns or occasionally over the cerebral hemispheres might produce a symmetric hydrocephalus. Exact localization of the point of obstruction may be quite difficult in these cases. It is important to realize that the mere fact that a certain structure does not fill readily does not necessarily mean that the tumor is in that location. It is very probable that encephalography conducted under ideal circumstances with the direction or supervision of a skilled neuroradiologist might add considerably to the localization of some of these lesions.

The third type of lesion as far as pneumography is concerned includes all cases in which the mass can be clearly recognized within the ventricles or in which the mass is so close to the ventricular system that it protrudes into the lumen and at least a part of the mass is clearly outlined by gas. In these cases the actual tumor is demonstrable, in contrast to the first group mentioned previously in which the diagnosis is based on the effect the tumor has had on the ventricles.

The fourth type of abnormality, and the least common, is seen when the gas actually enters into the tumor itself. This usually occurs in cystic tumors which have broken through into the ventricles or into the subarachnoid spaces and a free communication develops. In such instances the localization of the lesion is quite easy due to the fact that these abnormal gas patterns are quite readily recognized.

In recent years, using more precise technics of encephalography, tumors in the posterior fossa have been well demonstrated. In expert hands it is possible to locate most of the lesions in the posterior fossa which are projecting into the basal cisterns, such as the tumors of the pons and medulla and acoustic tumors.

In the centers where pneumography has been utilized most extensively and accurately, 90 to 95 per cent of all intracranial space-taking lesions can be localized.

ANGIOGRAPHY

Cerebral angiography is the most recent development in roentgen methods for the localization of intracranial lesions. Possibly the greatest contribution of cerebral angiography has been the demonstration of vascular lesions such as aneurysms, vascular thrombosis and arteriovenous malformations. Prior to the advent of angiography these lesions were not demonstrable by x-ray methods. It is also

possible, however, to localize a great number of brain tumors by angiography, and in many instances angiography is a preferable procedure to pneumography. The diagnosis of brain tumors by angiography depends on two factors: (1) displacement of the blood vessels and (2) demonstration of the characteristic vascular pattern of the neoplasm itself.

Localization by vascular displacement is dependent upon a thorough knowledge of the normal vascular anatomy of the brain as well as the many normal variations. There are many displacements and deformities of blood vessels secondary to neoplasms which are absolutely diagnostic in their localizing value, and in many instances this evidence may be more accurate than the findings observed on pneumography. There are also many instances in which the changes observed by angiography are equivocal and more accurate information might be obtained with pneumography. Unfortunately it is not always possible to predict beforehand which method will give the most reliable results.

In the second group are those lesions in which the characteristic vascular pattern of the tumor not only enables one to localize the lesion but also in many instances will indicate the histology of the tumor. The two types of lesions which often produce the most characteristic vascular pattern are the meningiomas and the glioblastomas. In both of these instances the tumors show an increased vascular pattern with abnormal vessels. The glioblastomas usually have an increased rate of flow of contrast media through the tumor into the veins. There are other tumors, such as the astrocytomas, which have a diminished vascularity. Some of the meningiomas receive a part of their blood supply from branches of the external carotid artery; and when this can be demonstrated, one can be sure that the lesion is a meingeoma since no other intracranial tumor receives its blood supply from the external carotid artery.

It is definitely possible to have a normal or relatively normal angiogram in the presence of an intracranial tumor, particularly those tumors which are intraventricular or near the base of the skull involving the brain stem. In actual practice, posterior fossa lesions are also difficult to demonstrate unless one is skilled in taking vertebral angiograms which seems to be a more difficult procedure than a carotid angiogram. At the present time it is probable that 70 to 80 per cent of intracranial tumors can be located by properly executed cerebral angiograms.

FALSE POSITIVE FINDINGS

A few remarks should be made concerning false positive roentgen findings. Anyone practicing neuroradiology should be impressed with the need for avoiding false positive x-ray diagnoses. This applies to all branches of neuroradiology including interpretation of the plain films as well as the pneumograms and angiograms. Although this hazard applies to all branches of radiology, it is more significant in neuroradiology. A false positive finding on the plain films may be the major factor in initiating angiography, ventriculography or encephalography, all of which are relatively serious procedures and not to be undertaken lightly. A false localization of a lesion on encephalography or angiography may result in exploration in a patient who has no tumor or exploration of the wrong area. These, of course, are very serious errors and must be avoided. Consequently, the reliability of the roentgen observations must be constantly weighed and the roentgen diagnosis carefully correlated with the clinical findings. In those instances in which there is doubt regarding the accuracy of the x-ray findings, any further procedures undertaken for diagnosis or treatment should be based primarily on the clinical indications rather than on atypical or unreliable roentgen findings.

CONCLUSIONS

1. Approximately 15 to 20 per cent of all brain tumors can be localized on the plain films.

2. Pneumography may be correct in 90 to 95 per cent of brain tumors when correctly utilized.

3. Angiography may be correct in 70 to 80 per cent of all intracranial tumors.

4. The radiologist should be ultraconservative in positive diagnoses so that false positives will be less than 5 per cent.

5. One can never absolutely exclude a brain tumor of any type by any x-ray procedure.

6. The earlier in the course of the disease that the x-ray examinations are performed, the greater the possibility of negative findings.

7. The value of the x-ray examination is

inversely proportional to the clinical experience and judgment of the referring physician.

DISCUSSION

JOHN A. CAMPBELL (Indianapolis, Ind.): Today the demands made on radiologists by neurosurgeons are high, and they are getting higher all the time. The neurosurgeons want the largest x-ray room, the best machine, the best technician, a separate dark room for their own films and the fastest service. The odd thing about this is that they probably deserve to have it. The field they are in, I am sure, has enough frustrations without adding ours to them. I think, in addition to this, they need exceptionally good radiologists to interpret films and give an independent radiologic evaluation. While we are not all as capable as Dr. Peterson, I think we should try to do the best we can. We should supply the technical needs that the neurosurgeon must have and try to add whatever we can in the way of interpretative skill. At the same time the radiologist must make a few demands on the neurosurgeon. He must understand that he cannot use the entire department and tie up some of our rooms so as to create too great a problem with the rest of our practice.

I think Dr. Peterson used the following original aphorism a few years ago with regard to localizing brain tumors, "Well, when I see a little something wrong, I call it normal." This is not a bad example to follow although I know that it, too, can lead to trouble. Probably the radiologist's main function is to keep both himself and the neurosurgeon from over-reading the films and jeopardizing the patient.

Electroencephalographic Diagnosis of Brain Tumors*

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From the Section of Physiology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

THE electroencephalogram (EEG) is now so commonplace and a shiny chrome-plated machine appears so often in physicians' offices that it is still insufficiently realized that electroencephalography remains a difficult technic. Full realization of the difficulty of this procedure and careful attention to certain technical points are essential if reliable diagnoses are to be established by this means. The most important requirements for satisfactory diagnostic recording are a skillful and well trained technician, a well trained electroencephalographer and a reliably serviced EEG instrument with eight-channel recording. The technician should have had a year of training in the technic and the interpreter at least six months; if these two requirements cannot be fulfilled it would be better to have no EEG examination since it can be a most dangerously misleading technic in the hands of inexperienced operators.

Many a misdiagnosis of tumor results from the presence of pulse or electrode artifacts, so that particular attention should be paid to the application and proper filling of electrodes. Eye movements should be carefully watched and controlled by holding the eyelids manually since these produce very troublesome slow-wave artifacts in the frontal and temporal regions. Sleep recordings are contraindicated, since not only do sleep rhythms mask delta foci but also the foci themselves sometimes disappear during sleep. An adequate number of scalp electrodes must be used (at least fourteen), and bipolar technic gives greatly improved diagnostic sensitivity over monopolar technic owing to the increased amplitude of out-of-phase signals and the cancellation of diffuse rhythms unimportant in tumor localization.

When the mechanism by which a tumor can influence brain rhythms is considered, it should be remembered that all such lesions are electrically inactive. An early effect of their presence is the production of interference with the normal cortical rhythms, often resulting in partial or complete suppression of the alpha rhythm on the side of the lesion. As the tumor continues to increase in size, its local irritative effects become predominant and slow delta waves result by a mechanism analogous to that cartooned in Figure 1. However, tumors do not always give rise to recordable local effects. Those lying deeply are more likely to produce their effects by irritation of pathways conducting toward the cortex. Thus bilateral rhythmic disturbances result, particularly from tumors situated near the midline in the regions of the third ventricle, brain stem and posterior fossa. Hence there is more neuronal organization behind these distant rhythmic effects, the mechanism of which is represented in cartoon fashion to the left in Figure 1. Since lesions produce their effects indirectly through pressure on surrounding structures, it is important to note that the EEG gives no direct evidence concerning the pathologic nature of the lesion. However, more rapidly invasive lesions produce most disturbance and, therefore, delta waves of the highest amplitude. More chronic lesions produce less delta and usually more rhythmic discharge. Hence it is possible to use this

* Read at the Ninth Annual Symposium on Malignancy given at the Indiana University Medical Center, Indianapolis, Indiana.

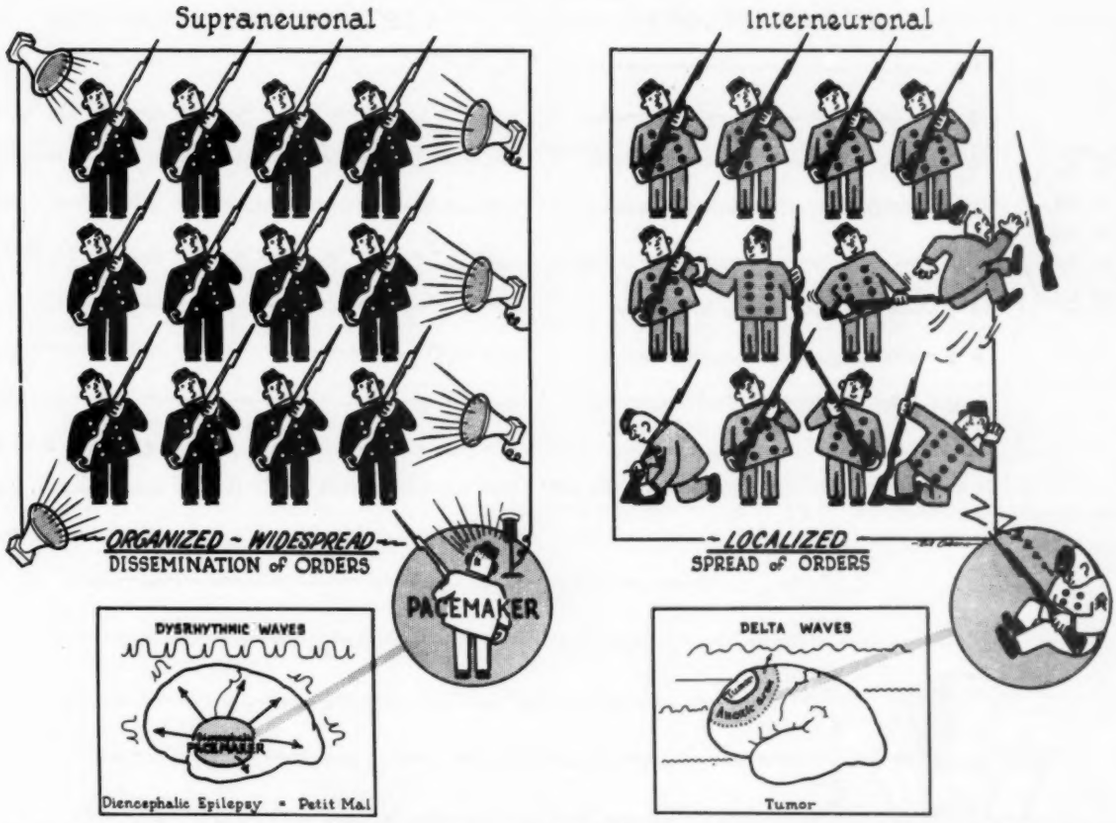

FIG. 1. Soldiers represent individual neurons. On the right, a local neuronal disturbance with hyperexcitability and interaction is represented. On the left, the neurons are under the influence of a diffuse synchronizing system, representative of what can happen when a deep or midline tumor causes irritative effects that are conducted along neuronal pathways.

indirect evidence concerning the pathologic nature of the lesion.

EEG CHANGES BY TYPE OF TUMOR

With these thoughts in mind, I shall review the EEG findings in three groups of brain tumors, namely, cortical tumors, subcortical tumors and midline tumors. In the last-mentioned category are included parasagittal, third ventricle, mid-brain and posterior fossa tumors, since these are likely to produce similar changes in the EEG. The EEG gives its best localizing results with cortical tumors since these are readily accessible to recordings by scalp electrodes. In collecting the data that follow I have had help from many workers in our laboratory, including Drs. Philip White, Donald Klass and David Daly. In a recent analysis of data on 100 cortical tumors, it was shown that 96 per cent of the tumors caused abnormalities in the recordings. Additional analyses disclosed that 87 per cent were focal; 76 per cent showed delta foci, all of which were correctly localized, and 11 per cent showed focal dysrhythmias, of which 10 per cent were correctly lateralized and 1 per cent was incorrectly lateralized.

The mechanisms discussed are represented in the following illustrations from actual cases. In Figure 2 the slow delta rhythms produced by a left frontal meningioma are compared with normal rhythms on the left. Meningiomas, being in the group of slowly progressive tumors, are among the most difficult to localize by EEG means. The parasagittal tumors are the worst in this respect. Figure 3 represents a right frontal delta focus in a nine year old child who had a right frontal astrocytoma. Since the normal rhythms are slower in a child, tumor diagnosis may be somewhat more difficult, particularly in view of the fact that midline tumors giving bilateral disturbances are common at this age.

Tumors do not always give rise to delta foci.

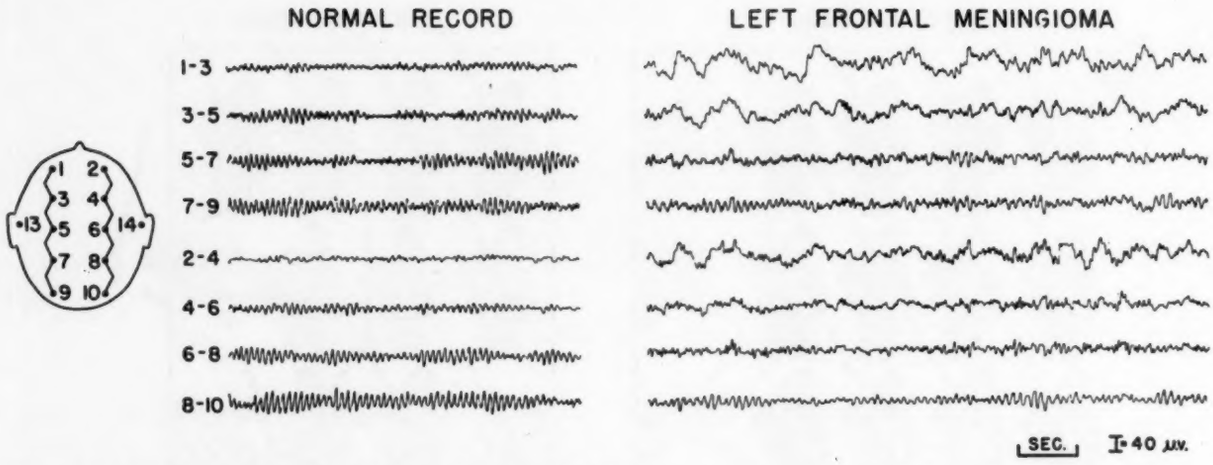

FIG. 2. Record from a normal subject compared with that from a patient with a left frontal meningioma. Delta waves are present in channels 1, 2 and 5.

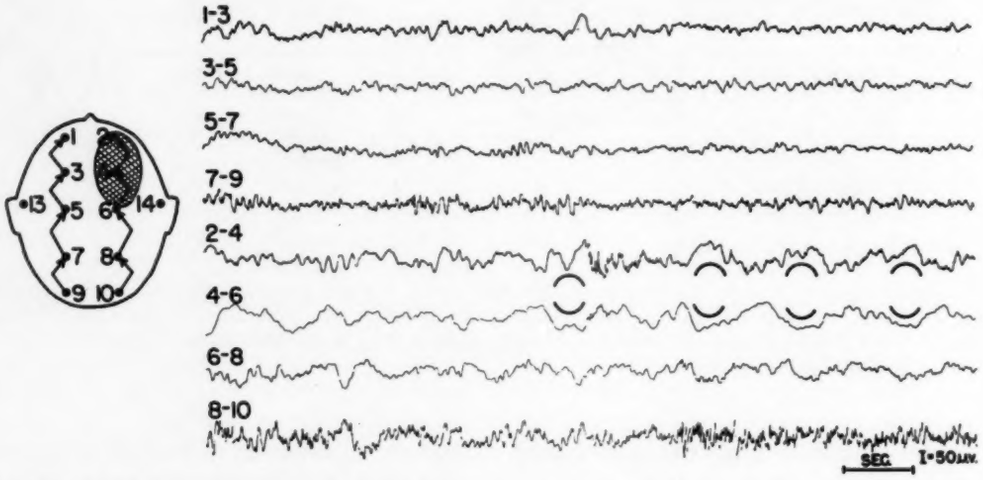

FIG. 3. Delta waves appearing in the right frontal region are underlined in black. This recording is from a nine year old child who had sustained an injury of the right temporal region two months previously, had had left hemiparesis for one month, and had bilateral choking of the optic disks. Operation disclosed an astrocytoma of the right frontal region.

The more slowly progressive kind may produce spike foci and thus are in danger of being mistaken for the chronic type of scar or atrophic lesion. Figure 4 represents an example of left frontal spike focus occurring in a patient who had a left frontal angioma with Jacksonian seizures. Similarly, metastatic tumors not infrequently give rise to discharges containing mixed delta and dysrhythmic components.

A tumor that occupies a subcortical position is more likely to affect midline structures and produce bilateral disturbances. A tumor of the posterior fossa may produce a similar effect by hydrocephalic expansion of the third ventricle. A good example of this effect is represented in Figure 5, which shows the bilateral frontal rhythmic disturbances resulting from a left cerebellar astrocytoma. It shows the remarkable change that has taken place in the EEG made eight months after removal of the tumor when the rhythms have returned to normal range. A feature commonly present in the projected type of bilateral disturbance is sensitivity to alerting, which may cause partial or complete disappearance of all abnormal rhythms for a time after stimulation. In the unstimulated patient, the recording may show a characteristic waxing and waning of the rhythms. It should be emphasized, however, that these rhythms are not specific and in many instances they can be seen in the records of patients with convulsive disorders. However,

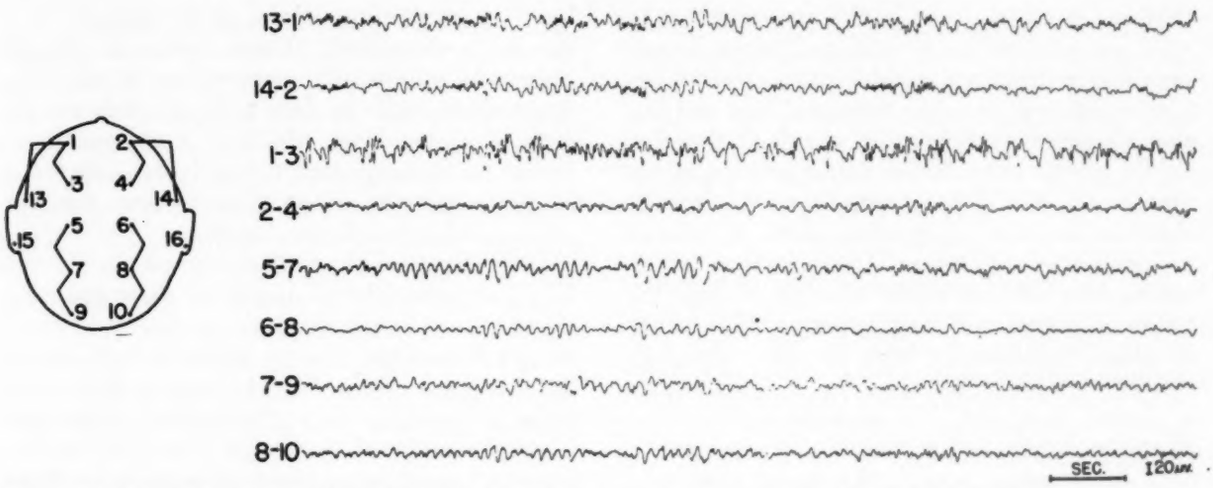

FIG. 4. Evidence of spike discharges is seen in the third-channel recording from the region of a left frontal angioma that caused Jacksonian seizures on the right side.

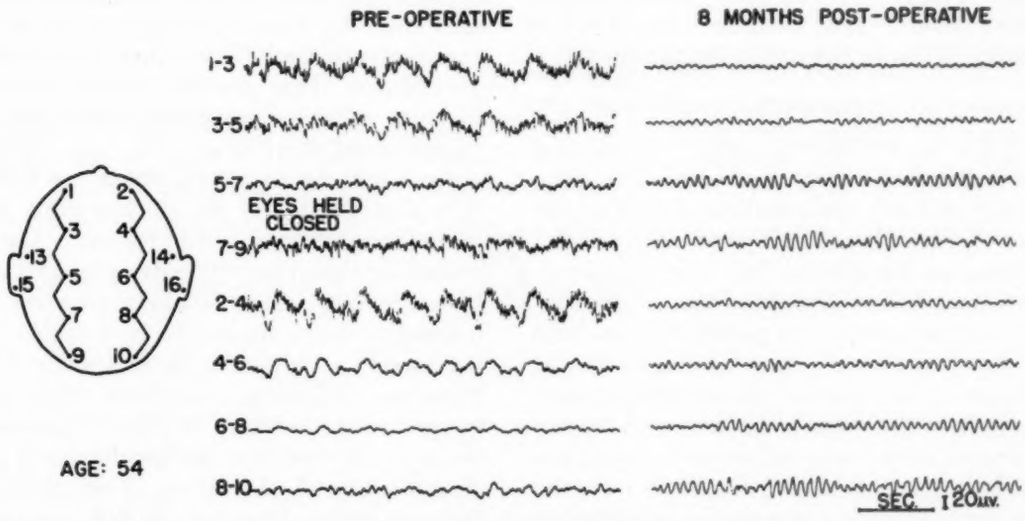

FIG. 5. Rhythmic waves with superimposed fast muscle spikes are evident in channels 1 and 5 on the left. These do not represent eye blinks. The postoperative change, represented on the right, is remarkable, and consists of restoration of alpha rhythm and disappearance of rhythmic bifrontal discharge. These recordings are from a patient with cystic astrocytoma of the left cerebellar hemisphere.

given the information that the patient has papilledema or that, from other evidence, he may be presumed to have a tumor, the occurrence of bilateral rhythms at once suggests that the tumor is deep and near the midline rather than in the cortex.

In summary of the results obtained in the presence of deep midline and posterior fossa lesions, I would like to emphasize the occurrence of bilateral rhythms responsive to alerting, but to observe that this type of abnormality may not develop very early in the course of the illness and that some 40 per cent of patients who present with symptoms suggestive of a midline lesion and who subsequently are proved to have such a lesion are found to have normal rhythms. It is obviously important, therefore, not to regard a negative result on EEG examination as in a sense ruling out a lesion in this region in the way it tends to rule out a cortical lesion.

In general, most activation methods are not very useful in the localization of tumors. Hyperventilation, however, frequently gives information of value and should be carried out in all cases, since it may reveal a delta focus

that is not clearly seen in the resting state. In addition, seizures can be initiated during hyperventilation in an appreciable proportion of patients with tumors of the temporal lobe and further diagnostic information obtained thereby. While photic stimulation often reveals asymmetries in the driving response, the results obtained are not, in general, quite as reliable an indicator of lesion lateralization as is asymmetry of the alpha rhythm. When the lesion is in the midline a paroxysmal response to photic stimulation may be seen, although this cannot be relied upon for diagnosis since it occurs frequently in convulsive disorders. Pentylenetetrazol (metrazol®) activation is only occasionally useful, this being true particularly when a focal seizure is induced. It has been found, however, that negative metrazol activation cannot be relied upon to exclude an expanding lesion. The frequency analyzer is only rarely of use in the diagnosis of tumors.

POINTS IN DIFFERENTIAL DIAGNOSIS

The EEG is particularly useful in screening cases of headache for organic lesions. The large percentage of focal abnormalities found in the presence of cortical tumors is particularly useful in this respect if a cortical lesion is suspected. Bilateral and diffuse abnormalities of rhythm such as may be seen in the presence of midline lesions are not, however, so useful diagnostically since about 15 per cent of the normal population may show this type of pattern and it may be associated with many other neurologic conditions. In a patient having focal seizures, the EEG is likely to contribute useful information in differentiating a tumor from a scar or atrophic lesion, since in the former a delta focus may be expected and in the latter a focal spike or other type of discharge is more commonly encountered. On the other hand, the existence of a spike focus does not exclude a tumor, and most of those who perform cortical excision of epileptic foci have at some time or other operated with the expectation of discovering a scar lesion only to find a tumor. The EEG has an important application in those patients who present with a convulsive disorder with some focal features and who, the clinician believes, may possibly have a tumor but whose present condition does not justify additional studies (pneumogram and so forth). Such patients are usually treated with anticonvulsants and even though they do, in fact, have an expanding lesion, their seizures may be well controlled. These patients should always be monitored by recordings at monthly intervals in order to detect the possible expansion of a neoplasm which in most instances would be accompanied by an increasing focus of delta activity since these waves are not suppressed by anticonvulsants.

In patients with confusional psychosis the EEG is particularly useful in distinguishing those whose condition may be due to a focal lesion. A number of these patients turn out to have tumors of the frontal lobe, and in such patients the EEG may give the first indication of focal disease of the brain. The EEG is frequently useful in patients presenting in coma without localizing signs. While diffuse rhythms are nearly always prominent in such patients because of coma, those in whom the symptoms are caused by a localized lesion such as a tumor are likely to show a localized delta discharge in addition. This is often clearly recognizable in spite of the disorganized background due to projected rhythms.

As might be expected, there are difficulties in using the EEG to differentiate between vascular and neoplastic lesions. The initial recording taken shortly after the development of a vascular lesion is likely to show an abnormality quite similar to that which may be produced by a tumor in the same location. However, follow-up recordings taken in three to six weeks are likely to show improvement if the lesion is vascular, but no change or perhaps deterioration if the lesion is neoplastic. This differentiation, however, is not 100 per cent reliable, since expanding lesions may occasionally be characterized by fluctuation in the EEG pattern.

A difficult question which the electroencephalographer may be asked is how to differentiate between recurrence of a tumor and development of a scar lesion in a patient who has undergone removal of a tumor and is now having focal seizures. In these instances, there is frequently a mixture of slow-wave and spike activity in the affected region. If slow activity is predominant, recurrence of the tumor is likely, whereas a predominant spike or rhythmic discharge favors a scar process.

A number of conditions give rise to difficulty in tumor diagnosis. The most important of these is probably the postictal phase of a seizure in which, to the uninitiated, the EEG

may suggest a focal or midline expanding lesion. However, a recording taken a day or so later may show that the slow waves have disappeared. Therefore, one should never trust recordings made shortly after a generalized or focal seizure.

Medication may produce important changes in the EEG, with prominent diffuse rhythms much like those resulting from lesions of the posterior fossa or from other midline lesions. Therefore, one must be on guard for the barbiturate addict with ataxia, confusional syndrome and evidence of bilaterally synchronous discharges in the EEG, lest drug intoxication be mistaken for a subtentorial lesion. Electroshock treatment may produce much bilateral discharge as evidenced in the EEG, usually maximal in the frontal and temporal regions. This may persist three to six months following the last shock treatment.

SUMMARY

The EEG is a screening method for organic focal lesions, particularly for cortical tumors, and to a lesser extent for subcortical and midline expanding lesions. With care in technic and interpretations, the neurologist and neurosurgeon are provided with a very useful and reliable method to supplement their clinical impressions.

DISCUSSION

PHILIP WHITE (Indianapolis, Ind.): The only thing I would like to add to Dr. Bickford's paper is to state that the EEG is a screening device which is safe, reasonably comfortable and painless. It has its applications and limitations. These must be known to the electroencephalographer as well as to the clinicians who use the service. I should like to re-emphasize the importance of technically adequate records, free from artifacts. I do not think the EEG competes with any of the neurosurgical procedures. We have friendly competition in discussing which is the most useful—angiography, pneumoencephalography or electroencephalography. The EEG has the advantages of being easy and safe, and it can be used repeatedly. It often is helpful in determining what other procedures are necessary.

Isotope Localization in Brain Tumors*

WILLIAM T. PEYTON, M.D., *Minneapolis, Minnesota*

IN September, 1947, we began the injection of radioactive iodine intravenously, followed by a survey of the head with a Geiger-Müller tube, to locate brain tumors. It was found that there was a differential uptake of the sodium diiodofluorescein, this material being taken up in greater concentration by the tumor than by normal brain tissue. This greater uptake by the tumor is apparently due for the most part to disturbance of the blood-brain barrier by the tumor or absence of a barrier in the tumor, but to some extent it is apparently also due to a greater quantity of radioactive blood within the tumor. We had some success in localizing brain tumors by this method. In the first twenty-six patients examined eighteen were harboring tumors, ten or 56 per cent of which were correctly localized.

The accuracy of this method depends on many factors. In any analysis by random sampling, the significance of the sample depends on the number counted. The counts can be increased in this head survey by increasing the time the tube is held over each position counted or by counting a larger part of the energy emitted over a shorter period of time.

The time during which counts can be made over an area is limited by the time a patient will hold his head completely motionless, and many patients with brain tumor will not do so for longer than a few minutes at most. Hence most counts have been made for a period of one minute.

More of the energy emitted can be counted by using a detector that registers the emitted energy from a wider field, that is a more "widely open" counter or one that sees a wider field. This is accomplished by reducing the collimation of the window of the counter. However, doing so reduces the directional qualities or accuracy of localization. We at first used a Geiger-Müller tube with a mica window 2.5 cm. in diameter and with a shielding lead cone extending 7 cm. beyond the window.

Later we used a tube with the mica window at the end of the tube that is without collimation. Such a tube is most effective in detecting tumors near the surface; it is also useful in a preliminary survey, to be followed by a second survey with a tube of better directional characteristics. The best tube is one that compromises between a high counting rate and one that has good directional characteristics but lower count.

Higher counting rates may also be attained by increasing the efficiency of the detector. The scintillation counter is much more efficient than the Geiger-Müller tube. Only 2 per cent of the gamma radiations of I-131 entering the gas-filled Geiger-Müller tube are detected, but the solid scintillating crystal of the scintillation counter may detect as much as 60 per cent of these gamma radiations.

For the past several years we have been using a scintillation counter partially collimated so that it covers approximately 15 degrees from and around the center of the cone.

This sectional cone of course is a section of the whole head extending from the point of contact across the head to include the skin of the opposite side. More of the volume of this cone lies on the opposite side of the midline, but this is more than compensated for by the fact that radioactivity decreases as the square of the distance. Nevertheless this area of the cone beyond the midline is an important factor in decreasing the percentage difference

* Read at the Ninth Annual Symposium on Malignancy given at the Indiana University Medical Center, Indianapolis, Indiana. This paper is a résumé of isotope encephalometry at the University of Minnesota and includes work of George E. Moore, M.D., PH.D., now Director of Roswell Park Memorial Institute, Buffalo, N. Y.; Lyle French, M.D., PH.D.; Shelley Chou, M.D.; Gerald Haines, M.D.; and Ralph Suechting, M.D.

between the counts on each side between two symmetric positions when one is depending on asymmetric counts for the localization of an intracranial lesion.

In our earlier attempts to localize brain tumors by radioactive isotopes we depended on finding a difference in the counts in symmetric positions on each side of the head, and a difference of 10 per cent or more was considered to be significant if it persisted on recounting of these positions.

This asymmetry may be focal or diffuse and this is to some extent correlated with the underlying pathologic condition of the brain. Focal asymmetry usually gives a 10 per cent difference in more than one adjacent position and little or no asymmetry in the surrounding areas. It is prone to occur over a superficial lesion without edema in the surrounding brain. Diffuse asymmetry gives a 10 per cent difference often only in one area but with a decreasing difference in the other positions on the same side so that the exact margin of the asymmetry is difficult to define. This diffuse asymmetry is prone to occur when the lesion is deep or is surrounded by edematous brain tissue.

In October, 1950, about the time we began to use the scintillation counter, we also began to use radioactive iodinated serum albumin RIHSA* instead of diiodofluorescein DIF*. Because RIHSA* gives a sustained and therefore stable blood and tissue level, it has come to be the tracer material of choice for isotope encephalometry. In one hour after intravenous injection of DIF*, radioactivity in the blood stream drops to 50 per cent of the initial level, whereas twenty-four hours after the injection of RIHSA*, radioactivity in the blood is still approximately 60 per cent of the original level.

Therefore when DIF* is used, counting is begun approximately one hour later and symmetric positions must be counted on each side as the head survey progresses because there is a significant drop in the counting rate during the period required for a complete survey. Repeat examination or rechecks of suspicious areas must be done immediately and even then are less accurate because of the lower counting rate.

When RIHSA* is used, the first count can be made as early as one hour after the tracer material is injected. However, usually the first count is made twenty-four hours later when a greater differential uptake exists between the tumor and normal brain; repeated counts can be made again in the next twenty-four to forty-eight hours, during which time the counting rate drops very slowly but the differential uptake between the tumor and normal brain may even increase. The only disadvantage in using RIHSA* is that it is gradually metabolized liberating free I-131 and this is taken up by the thyroid unless the thyroid is previously blocked by the administration of stable iodine.

A group of fifty-seven cases, counted with an improved tube and using DIF* as a tracer medium, was analyzed. In this group there were twenty-six tumors, seventeen or 56 per cent of which were correctly localized.

We tried to be as objective as possible in making the diagnosis by the isotope study in these patients. The patients' clinical records were not utilized, but even so it was inevitable that the examiner was influenced by physical signs which were obvious, such as aphasia or hemiplegia. To evaluate this factor, these counting records were again examined and interpreted by the same person who made the original interpretation but in this second survey only the data were available. On this second survey of fifty-seven cases five localizations that were previously correct were now wrong.

Another group of forty-six cases was analyzed. These cases were counted with a scintillation counter and RIHSA* was used for the most part as the radioactive tracer. The counting records were interpreted first with all the clinical data but without angiography or air studies, and then interpreted again with only the counting data available.

In this group there were seventeen tumors, all of which were correctly localized when interpretation was made with the aid of the clinical record. However, when interpreted blind from the data five tumors were not localized, that is, the accuracy of localization dropped from 100 to 71 per cent when the clinical data was not utilized.

It appeared that some method of analysis of the data other than by asymmetry might improve the accuracy of localization. Midline lesions naturally could not be detected by asymmetry, and cystic lesions which took up less radioactive material than the normal brain

* Signifies radioactive.

University of Minnesota Hospitals
Isotope-encephalometry

Hosp. No..... Name..... Age..... Wt..... BG..... Date.....

Injection time..... Tracer.....
Counting started..... Counter.....

Area	Right	Left	%D	Area	Right	Left	%D	Area	Right	Left	%D
1				1				1			
2				2				2			
3				3				3			
4				4				4			
5				5				5			
6				6				6			
7				7				7			
8				8				8			
9				9				9			
10				10				10			
11				11				11			
12				12				12			

FIG. 1. Form used in recording results of isotope encephalometry. It illustrates the numerical positions at which counts are made.

would tend to give a false diagnosis of a focus on the wrong side.

A graphic method of analysis was therefore developed. The normal activity for each position of the head and its relation to the other positions was determined. The data from twenty-seven people whose history did not suggest that they had an organic lesion and in whom examination including air studies and angiograms did not reveal a lesion were utilized to obtain a normal curve of radioactivity. DIF* with its unstable counting rate is of course not suitable for this type of study. These twenty-seven persons were each given 5 μc. of RIHSA* per kg. of body weight and counting was done with a scintillation counter. A sum of all these counts and a sum of all counts for each position were calculated, and then the average total count for each case or 260,000 counts per minute of counting and similar average for each of the twenty-four positions counted were determined. Since they were essentially the same on each side, actually only twelve such averages were computed utilizing the sum of the counts on each side or fifty-four countings to determine the average for each position. A normal graph was then made for each side by plotting these average counts for each of the twelve positions against each position arbitrarily arranged in series from position 1 to position 12. (Fig. 1.) In this graph counts for positions 2, 5, 8, 11 and 12 are all high because

they are near the base of the skull where muscle lies between the counting tube and the skull. Positions 1, 6 and 9 are the lowest counts because they are made over areas where only scalp intervenes between the tube and skull. At the same time the tube is not directed at a background of high activity as is the case in positions 4, 7 and 10, where also only scalp intervenes between the tube and the skull but where the tube is directed at the base of the skull with its associated mass of soft tissue.

TABLE 1

	Asymmetry	Graphic Method
Tumors correctly localized	17 (68%)	21 (84%)
Tumors not correctly localized	8 (32%)	4 (16%)
Other space-displacing lesions correct	5 (71%)	7 (100%)
Other space-displacing lesions incorrect	2 (29%)	0
No lesion or focus localized	36	36
No lesion but false focus localized	1	1

Since the counting rate will vary greatly even with a constant dose of radioactive material and also the time of counting, etc., kept constant, in order to compare the curve of each unknown case with this normal curve it is necessary to convert each to the scale of the normal by multiplying by a factor determined by calculating the ratio between the sum of all counts (260,000) of the positions in the normal and the sum of all the positions in the case under analysis. Interpretation by this graphic technic depends primarily on the shape and slope of the curve in question when compared with the normal graph.

During the past year sixty-nine patients were given RIHSA* and counts were done with a scintillation counter. The data were analyzed by both asymmetry and the graphic method. They were analyzed without clinical or other laboratory data. The results are shown in Table 1.

CONCLUSIONS

Isotope encephalometry is an innocuous procedure which can be done without discomfort to the patient.

If a positive focus is found, it is quite certain that the patient has a tumor, abscess, hematoma or other gross cerebral lesion. A negative finding, i.e., no focus demonstrated, is far less reliable.

If utilized with the clinical data, isotope encephalometry is quite accurate and useful in selecting doubtful cases for additional study by air injection or angiography.

If those cases in which a good focus is not demonstrated by asymmetry are analyzed by the graphic method, the accuracy of results will be significantly increased.

We have rarely depended on isotope localization alone for the location of a bone flap.

DISCUSSION

JAMES G. LORMAN (Ft. Wayne, Ind.): The preceding presentations have demonstrated to us that in addition to the neurologic examination the neurosurgeon has several valuable aids at his disposal. These aids, as a group, give a high degree of accuracy in determining first if a tumor is present and second where it is located. Dr. Peyton's presentation is of particular interest because he is utilizing physiologic tumor metabolism as an external means of tumor detection. With the information arrived at by the aforementioned means the neurosurgeon can plan an accurately localized craniotomy. When the cortex is exposed, most tumors present directly or by vascular and/or convolutional changes. Sometimes, however, when these signs are absent it becomes necessary to explore tissue underlying the cortex to determine the exact location and extent of the tumor. Radioactive isotopes can also be used for this type of localization. In 1951 Silverstone and White recorded a series of cases in which a small dose of radioactive isotope was given prior to surgery. At surgery the isotope was detected in the neoplastic tissue by means of a small needle-like probe containing a Geiger counter. The neurosurgeons with whom we worked encouraged us to include this service in our isotope program. The following procedure was evolved:

As soon as the time of a craniotomy for neoplasm is set, we plan to give a small dose of P-32 to the patient. If possible it is given twelve hours prior to surgery, although successful recordings have been made when the P-32 was given less than four hours before surgery. Our brain probe has a diameter of approximately 2 mm. It is attached to a count rate meter which has an audible and visual recorder. After the dura is exposed, the probe is introduced into the brain. An area of normal brain tissue is entered and a normal brain background count is obtained. The probe is then advanced into the cortex in various directions and

at various depths, the counts being recorded at each respective position. In tumors that take up P-32 there is a striking difference in the count rate as the probe passes from normal into abnormal tissue. This difference may amount to a ten- to fifteenfold increase, for example from a normal brain background of 100 counts per minute to 1,000 counts per minute in tumor tissue. There is a striking difference between normal and tumor tissue due to the relative short penetration of P-32 beta emission. The procedure permits the surgeon to determine the site of the tumor quickly and to take a biopsy specimen without unnecessary trauma to the brain. It also helps to determine the extent of the tumor so that the surgeon can know when it has been completely removed.

Treatment of Intracranial Neoplasms*

PERCIVAL BAILEY, M.D.,† *Chicago, Illinios*

I HAVE enjoyed listening to this symposium although it is for the most part an old story to me—like listening to a song which was popular in your youth. I was first concerned with this problem in 1919 when I went to work with Harvey Cushing in Boston. At that time knowledge of intracranial neoplasms was very primitive. Cushing had just come back from World War I and progress had practically ceased during that war. You would have trouble today understanding the difficulties encountered at that time. We did not have any of the things which you talk about nowadays. The technique of surgical operations was quite limited.

You will remember that the first brain tumor, without any external signs of its presence was exposed in 1884 by Godlee, a surgeon of London. Tumors had been removed before that but there had always been some external sign, such as Dr. Peterson has shown you, an erosion of the skull, or an osteoma betraying the situation of the tumor. The operation of Godlee became understandably famous because the localization of the tumor was made, from clinical symptoms alone, by a neurologist named Bennett. Bennett was able to localize the tumor because another neurologist in London by the name of Hughlings Jackson and before him a physiologist in Germany named Hitzig had shown that a lesion developing in a given part of the cerebral cortex gave rise to symptoms which would point to its localization. On that basis Bennett localized the tumor in a part of the motor cortex and Godlee exposed and partially removed it. He did not completely remove the tumor; and it recurred shortly afterwards. The patient died from it, but the fact that it could be localized and exposed without any external signs made Godlee's operation the beginning of modern brain surgery. Shortly after this Victor Horsley exposed and removed a tumor in the spinal canal which had been localized by Gowers. This proved that Percival Pott was wrong when he said that a surgeon who would attempt to expose the spinal cord should be sued for malpractice. We date the beginning of modern surgery of the nervous system from those two operations.

The next great advance was made by Harvey Cushing. Horsley, of course, was a good physiologist but he was a rather crude surgeon. If you had seen him operate, splashing around in the operating room in rubber boots, the floor covered with antiseptic solution which came down from a big tank in the ceiling, you would recognize that it was not a very surgical procedure. Harvey Cushing applied to surgery of the brain the meticulous technique that he had been shown by his teacher—Halstead—and it was this advance which really made surgery of the brain possible.

Cushing generally gets credit for being the pioneer of brain surgery, which he was. Also he was thought to be a man with a great deal of temerity. This was not true—Dr. Cushing was an exceedingly sensitive person and rather timorous. He could not accept an operative mortality higher than 10%. If it went higher than that, he began to do less and take less chances for a while until the mortality dropped down below 10%. There were perhaps two reasons for his action—one was that he himself was quite sensitive and also he had a belief that the medical profession would not accept a procedure which carried a higher mortality, so he maintained very careful statistics and kept his mortality deliberately below 10%. This

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caused me some trouble when I first began to operate on my own in Chicago because the idea got around that he had removed all those tumors with a mortality of less than 10%. Dr. Peter Bassoe used to send me tumors, and when I could not remove them, he wanted to know "why?" I was a pupil of Cushing, and Dr. Cushing could remove such tumors with a mortality of less than 10%—why couldn't I? Well, at first, I was rather reluctant to tell him, because, you know, you are not supposed to criticize your teachers or your elders, but finally I had to tell him that Dr. Cushing had not removed all those tumors. What he had done very often was to make a small biopsy, and the statistics told some very erroneous things. All statistics have to be interpreted. You know the old gag about liars, damn liars and statisticians. Dr. Cushing's statistics proved very nicely that you could expose a brain tumor with a mortality of less than 10%, but it did not prove at all that you could remove such tumors with a mortality of less than 10%. When Dr. Bassoe understood that, he became rather hypercritical and began to criticize everything that was told him about intracranial surgery, particularly if it came from the Mayo Clinic. Before that he was very much inclined to think that the people at the Mayo Clinic were "tops." But after that he began to bring me articles from there and make all kinds of criticisms; some were justified and some were decidedly not justified. So I had my troubles in the beginning trying to keep people accurately aware of what was really going on.

If you ask me to tell you what percentage of tumors were removed successfully, it would be very small, indeed. I never have made any attempt to figure out the exact results. I should say that, in the case of intracranial meningiomas, maybe 20% of them were removed completely. In the case of the acoustic tumor, none was removed completely; they all recurred very shortly. In the case of adenomas of the hypophysis, none was removed completely, and most of them, of course, had a recurrence of symptoms before very long. In the case of suprasellar tumor, I know of only one that was removed completely, and that was largely by accident. In the case of the gliomas, almost never was one of them completely removed. A variable amount of tumor was removed in some cases. Most of them simply had a decompression with partial relief of the intracranial pressure. We have advanced a considerable distance since then for various reasons. In the first place, as you have heard, today we have much better methods for localizing these lesions. In those days, we were lucky if we could lateralize the tumor and tell whether it was in the right hemisphere or the left one. Nowadays, localization of this kind would not satisfy anyone. We want to know exactly where the lesion is, not only on which side it is, but also in what part of the hemisphere it lies. Even after an accurate localization is available, we can do much better than we used to because of advances in the technique of removing brain tumors. If you talk about meningeal tumors now, I think that you can safely say that 75% of them can be completely removed and a higher percentage than that can be almost completely removed.

We are able to stand much more serious complications than we could in those early days. We don't fear infection now as much as they did then. We don't fear hemorrhage as much as they did then. You haven't any idea what a transfusion was like in Dr. Cushing's operating room. It upset the whole hospital. The donor had to be brought into the operating room in order to get his blood into a paraffin-lined Kimpton tube. Often times it clotted before it could be given to the patient, while all the time Dr. Cushing was pacing up and down, fuming and making suggestions.

Methods of operating tumors in inaccessible situations were developed during my time in Cushing's Clinic. For example, a meningeal tumor on the surface, you could take out, but a meningeal tumor which lay on the base of the brain was almost inaccessible. The first successful attack on a meningioma at the base of the brain was made by using electrocautery, or the electrosurgical knife. This instrument was developed by a biophysicist in the Huntington Hospital, a hospital in Boston devoted to the treatment of cancer. I remember very well the attempts to get Dr. Cushing to use it in his operating room. Of course, after he first used it, he tried it on everything, and it did make it possible to take out certain tumors which could not have been taken without electrosurgery. Also when Dandy suggested that an acoustic tumor could be completely removed, Dr. Cushing wrote a nasty letter to the superintendent of Johns Hopkins Hospital and told him that Dr. Dandy should be stopped from making such ridiculous statements. Of course, Dr. Dandy just went on removing acoustic neurinomas. Up to that time Dr. Cushing had never

removed an acoustic tumor completely, and he did not believe that it could be done. Now, they are frequently removed completely. Another technical device which helped to make the removal of the intracranial tumor possible was the suction apparatus. I remember when Holman, who is now Professor of Surgery in San Francisco, first suggested to Dr. Cushing that he use a suction tube to keep the blood out of the way so he could see what he was doing. Dr. Cushing was indignant and said he did not wish to go down to posterity through a sucker. It was quite a while before he could be induced to use it. Of course, after he started to use it he would not be without it just as we can not be without it today. Everybody uses the suction apparatus, making it possible to remove a great many tumors we could not then remove, for example adenomas of the hypophysis.

Dr. Cushing had never taken out an adenoma of the hypophysis. He went through the nose, and he took out only what extruded spontaneously out of the opening in the sphenoid bone. Nowadays, the surgeons go in and take not only the tumor but the hypophysis also. We are able to do that because we now have hormone replacement therapy. I remember the first time I ever saw Dr. Cushing. He came to Chicago to a meeting where Frazier talked. Frazier reported a series of operations on suprasellar cysts, and said that he expected to live to see the day when those tumors were completely removed and to be able to present to that society a series of such cases. Dr. Cushing got up and said, "well, I should just like to point out that if Dr. Frazier's hopes were realized he would report exactly the same number of fatalities as he had patients, because the tumors are so intimately connected with the hypophysis that he could not possibly remove them completely without removing the hypophysis also." Fortunately, we don't have to worry about that nowadays because we have replacement therapies which can deal with the disturbances resulting from the removal of the hypophysis. So that we do much more complete extirpations now of hypophysial tumors than we could in those days.

Well, I have dealt with the meningeal tumors, with the acoustic tumors—I haven't told you what the mortality is—I don't know. I haven't been operating on tumors for the last five years—I am way behind the time, but perhaps we can say that 65% can be removed without killing the patient. In the case of hypophysial adenomas, we can do much better than that at the present time.

When we come to gliomas, however, the picture is not much better than it was in those days. The reason is that it is almost impossible to completely remove gliomatous tumors. They spread too widely in the nervous system. Even a hemispherectomy is not sufficient to remove a great many of them. They spread through the corpus callosum, they spread down the nervous pathways into the brain stem or the basal ganglia where you can not follow them without leaving the patient much worse off than he was before. Statistics vary as to the number of gliomas that have spread in this fashion before they reach the surgeon's hands. I should say that as many as 35–65% have already spread so far that they could not be removed without seriously injuring the patient. Those that can not be removed must be treated in other ways because the physician's business is not only to cure but also to alleviate the patient's symptoms, if he can. Therefore, other methods of treatment have been developed. The principal one is radiation, of various kinds. First of all, x-ray radiation, and more recently other types of radiation have been developed, ordinary x-rays or x-rays of a very high voltage with the beta electron or other forms of particles that are put out by the cyclotrons, cosmatrons, etc. These different forms of treatment have not proven to be too useful. They don't do very much with the most dangerous, the most fatal of the brain tumors, the glioblastoma. They produce good results on the medulloblastoma, but you can not effectively radiate the entire glioma without producing bad effects on the nervous system itself. To my knowledge no tumor of the glioma group has ever been cured by any radiation. I just came back from a meeting at Brookhaven, where I was not at all impressed with the results which they have obtained, and the expense, as Dr. Peyton said is staggering. It costs about $40,000 to treat one patient by the boron method. All they have succeeded in doing is to increase the life expectancy of such patients to maybe 50% over what it was with ordinary x-ray radiation. So we are not much farther along with the treatment of these gliomatous tumors. With the circumscribed tumors we have made real progress. A patient with such a tumor has now a much better opportunity of having his tumor removed and still be in condition to carry on an active life.

Experiences with Extracorporeal Circulation in Open Cardiac Surgery

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THE surgical efforts to replace the heart and lung by an extracorporeal circulation have been slow but have moved always in a forward direction. The perfection of such a by-pass is necessary, as many cardiac lesions can be corrected only by such an exclusion of the heart and lung. Lesions of the heart have been treated surgically in one of three ways. The *closed* method of heart surgery does *not* require direct vision of the inside of the heart. Such lesions as pericarditis, coarctation, patent ductus arteriosus, aortic-pulmonary window and aneurysms can be corrected. Some of the anomalies can be indirectly attacked by closed by-pass operations such as the Blalock or Potts operations for pulmonary stenosis. Included in the closed approach are the blind approaches to heart lesions such as the Brock maneuver for pulmonary stenosis, the blind fracture of the aortic valve by a dilator and the mitral commissurotomy. These are effective to some extent but the inability to visualize the proceeding detracts from the possible success. Such operations are makeshift affairs but in some cases have been most effective. (Table 1.)

TABLE 1
HEART LESIONS REPAIRABLE WITHOUT HYPOTHERMIA OR HEART-LUNG BY-PASS

1. Mitral stenosis
2. Minimal mitral insufficiency
3. Pericarditis
4. Pulmonary stenosis (Blalock, Potts and others)
5. Patent ductus arteriosus
6. Coarctation of aorta
7. Pulmonary-aortic window
8. Anomalies of aortic arch
9. Aneurysms

The second approach to heart surgery is an *open* heart technic using hypothermia. This modality allows the circulation to be stopped for a set, limited time. The strict time limit imposed by the latter, however, leads to hurried and sometimes incomplete surgical technics. Despite this restriction this method has led to the correction of pulmonary stenosis and atrial septal defects in many patients [13,14]. Since it will take a long time for all cardiac surgeons to be successful with extracorporeal circulation systems, hypothermia is advocated as an expedient to bridge the time gap until pump oxygenators are simplified enough and the cardiac surgeons are sufficiently experienced for this method to be a routine measure in all clinics. It is certain that innumerable patients have died waiting for the advent of this day.

The third method of heart surgery is by cardiopulmonary by-pass using a pump oxygenator. In the United States 50,000 children are born each year with congenital heart defects. One-half of these patients can be operated upon only with a heart-lung by-pass machine. To this number can be added the

acquired heart lesions, perhaps 40,000 new ones each year, not repairable without this method and those that can be treated more satisfactorily with the open heart technic.

Studies have brought out certain fundamental points which are basic in the consideration of any extracorporeal circulation method. These are:

1. Serious changes occur after oxygen lack for three minutes to the brain cells and ten minutes to the spinal cord. Brain cells die if deprived of oxygen for from four to five minutes.

2. These time limits can be lengthened by lowering the body temperatures. (Table II.)

3. The heart muscle cells also require oxygen supply and CO_2 removal to prevent their death. Hyperventilation of the lungs helps to prevent anoxia of the heart cells.

4. With an adequately functioning extracorporeal circulation the heart and lungs can be by-passed for an indefinite time. Open heart operations can be performed.

5. Such an extracorporeal circulation has inherent dangers such as cardiac ventricular fibrillation, clotting and embolism, derangement of the acid-base balance and of the metabolites and electrolytes, all of which must be controlled for success.

6. The heart can be stopped or placed in fibrillation during extracorporeal circulation for a period of fifteen to twenty minutes. This permits the completely quiet and dry heart desired for operation—again within a definite time limit. It is the hope and belief of all cardiac surgeons that this time interval eventually can be eliminated or lengthened sufficiently to make definitive surgery, under unhurried circumstances, routine.

Types of Heart-Lung Machines. Most cardiac pump oxygenators originated by adapting variations of blood transfusion apparatuses already in use. Our early efforts were along this line. In 1949 we equipped a direct transfusion apparatus of the Soresi type with a motor and utilized it to by-pass occluded major vessels. Later efforts to connect an oxygenation system to this machine were fraught with so many technical difficulties that the project was abandoned. The original work on heart pumps was premised on the belief that all the blood of the body had to be diverted and oxygenated before the heart could be opened. Many of the pumps still work on this principle. (Table III.) A little-noticed experiment by Andreason and Watson [*1*] changed this concept. They demonstrated that with the superior and inferior vena cava ligated, the blood which the azygos vein returned to the heart could keep the experimental animals alive for thirty-five minutes. This introduced the azygos principle, namely, that only part of the blood of the body had to be oxygenated to preserve life. (Table III.) On this basis many of the successful pump oxygenator apparatuses in use have been developed.

TABLE II
HEART LESIONS REPAIRABLE WITH HYPOTHERMIA

Pulmonary stenosis
Atrial septal defect
Some tetralogy of Fallot
Aortic occlusions

TABLE III
TYPES OF CARDIOPULMONARY BY-PASS

I. Methods of oxygenating all the blood:
A. Film oxygenator of the Gibbon type
B. Stationary oxygenator of the Gibbon type
C. Moving oxygenator of the Dennis type
II. Methods of oxygenating part of blood (azygos principle):
A. Bubble oxygenator pump (deWall-Lillehei)
B. Membranous oxygenator (Kolff-Balzer)
C. Variations in pumps or oxygenator apparatus

The cardiopulmonary machines in which the entire circulation is oxygenated while the heart and lung are by-passed (exemplified by the Gibbon, modified Mayo, Dennis and other machines [*2–6,12*]) as well as those in which only part of the circulating blood is oxygenated (such as the DeWall-Lillehei [*7,11*]) both work. After considerable observation we selected the partial oxygenation pump for our work. The main advantage to us is this method's simplicity in set-up and management. The ideal pump oxygenator of either system is not yet produced, and the one we have selected may have to be discarded at a later date. My purpose in this presentation is to perform a service which, I believe, is unusual. I wish to point out the difficulties to be encountered in performing operations on experimental animals with the extracorporeal (Fig. 1) pump oxygenator. Many who may try to use these pump oxy-

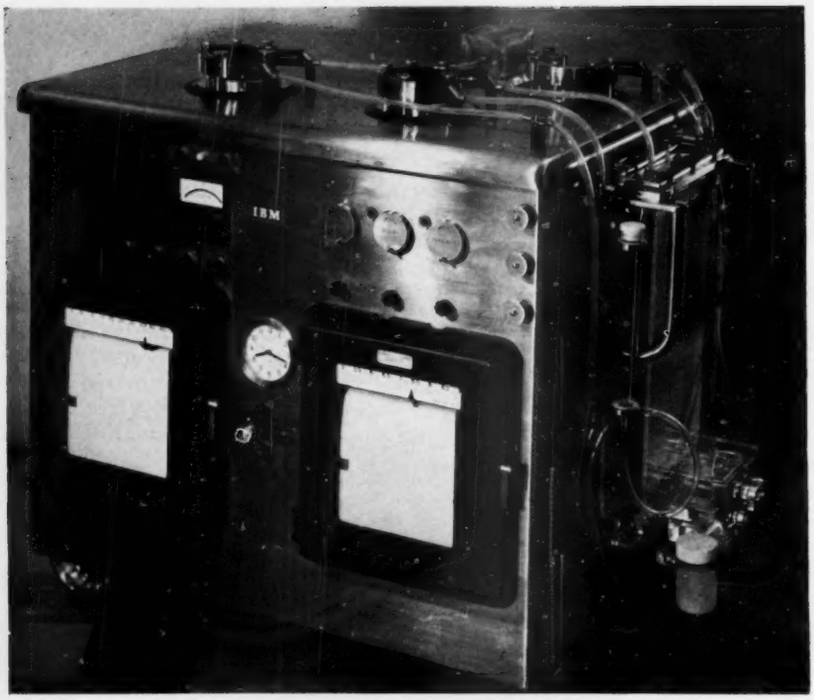

FIG. 1. The Gibbon pump oxygenator.

genators clinically have not spent the time and travel necessary to see them functioning in the cardiac surgery centers. The technical points which have to be surmounted in perform-

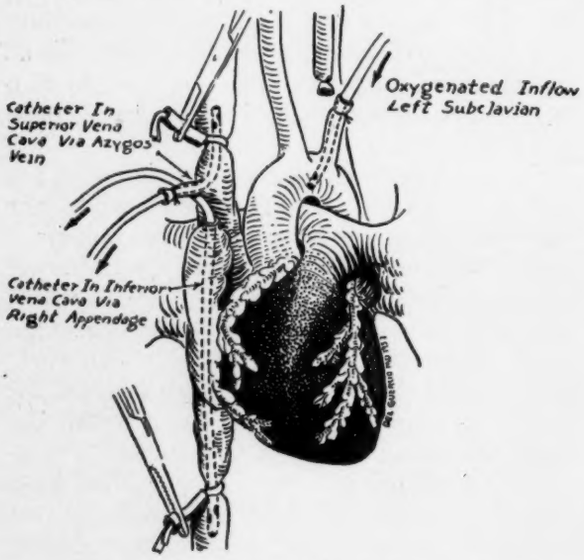
Catheter In Superior Vena Cava Via Azygos Vein
Oxygenated Inflow Left Subclavian
Catheter In Inferior Vena Cava Via Right Appendage

FIG. 2. Diagrammatic drawing of the intubation for by-pass of the heart and lungs.

ing successfully artificial heart-lung work require much experience. A discussion of the errors and difficulties which we and others have encountered in this work should be pooled for the information of all so that some of these problems may be avoided if possible.

In our work with this pump on animals we have learned many things. The most important was the length of time and the number of operations that were required before an animal survived open heart surgery. This time interval was necessary despite the fact that we began with a trained team experienced in all types of major and thoracic surgery. Our team* consisted of three cardiovascular surgeons, a full-time Fellow at the fifth-year level, a resident anesthetist, a graduate scrub nurse trained in thoracic surgery, and a regular surgical resident. To these were added a circulating nurse attendant, two senior medical students and two volunteer trained technicians. The departments of pathology and hematology cooperated in the blood chemistry, blood type crossing and hemolysis tests as well as in the autopsies. The cardiopulmonary unit ran our oxygen saturation pH and carbon dioxide studies. The dogs were given all night postoperative care. This team personnel is detailed only to emphasize the amount of work and number of helpers necessary to achieve success. (Fig. 2.)

* Other members of the Cardiovascular Surgery Unit are William Wolff, M.D., John Comer, M.D. and John Caputi, M.D. (Fellow).

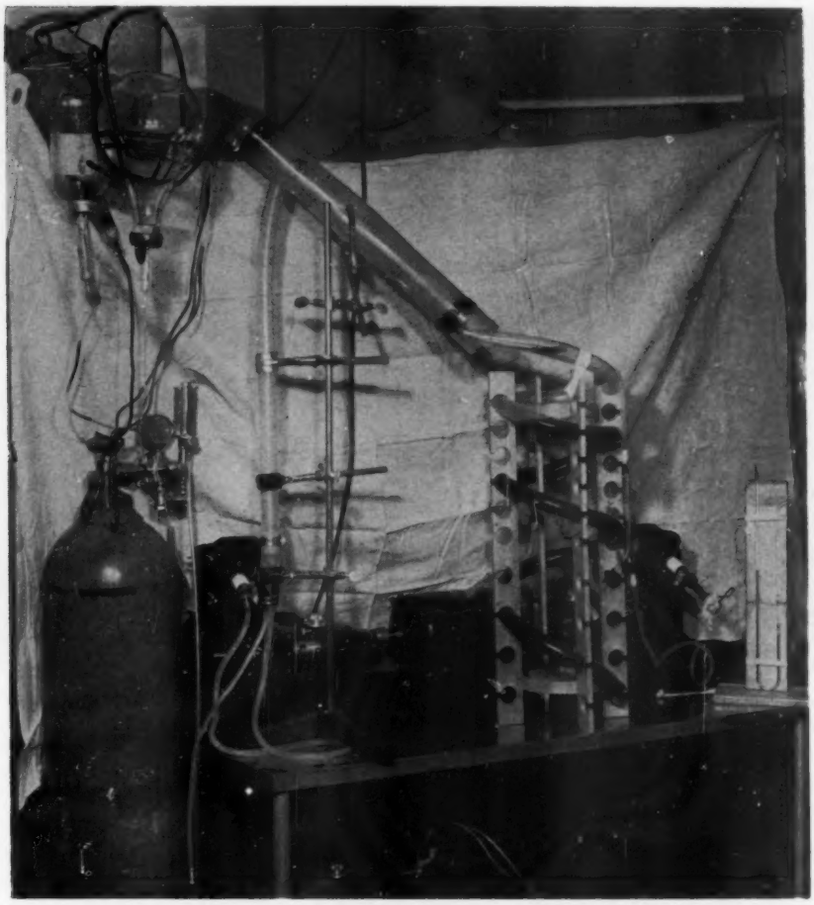

FIG. 3. Our own pump oxygenator in use (modified from DeWall-Lillehei). We now have doubled the size of the oxygenator.

With all in this group cooperating we performed two and three operations each week for five months before a dog survived the surgery.

It is not uncommon to hear of operations on animals being failures for so long that the surgeon finally has tried the operation on a human being despite his lack of success with the animals. It is difficult to imagine such an occurrence but it may have happened.

Principle of Partial Oxygen Pump (DeWall-Lillehei or University of Minnesota). The azygos principle pump permits oxygenation of approximately one-seventh of the body's blood supply. The azygos vein carries 10 to 18 ml. blood per kg. body weight per minute. Lillehei [7] and Varco et al. [2] reported that oxygenation of 35 to 45 ml. of blood per kg. body weight per minute resulted in the survival of nearly all the animals placed on the pump, provided there were no technical errors. To be as effective in artificial oxygenation on humans (Fig. 3) as he is on animals the surgeon has but to overcome these technical errors. There have been variations in this technic using the same partial oxygenation principle. Kolff and Balzer [9] use the same type of pump but utilize a polyethylene membrane to oxygenate it. They have now changed this technic to use a hydraulic compression through fluid medium as a pump and the oxygenator is now made of tethylon. Others, including our Clinic, have modified the procedure. (Fig. 4.)

With this heart-lung technic, lesions of the heart previously not corrected or only partly helped have now been operated upon successfully. These are: (1) ventricular septal defects (Fig. 5), (2) atrioventricularis communis, (3) isolated infundibular pulmonary stenosis and, (4) the tetralogy and pentology of Fallot.

To these can now be added some of the acquired heart diseases which kill so many persons. In the hopeful future are the surgical treatment for coronary artery diseases which is still in the stage of development (Fig. 6) and

FIG. 4. Operation on dog using total by-pass. A, shows tube through the right auricular appendage to cannulize the inferior vena cava. B, shows tube placed in the superior vena cava through azygos vein; note vaseline gauze used to occlude the cavas. C, catheter in the aorta through subclavian artery; oxygenated blood is returned to the body through this tube.

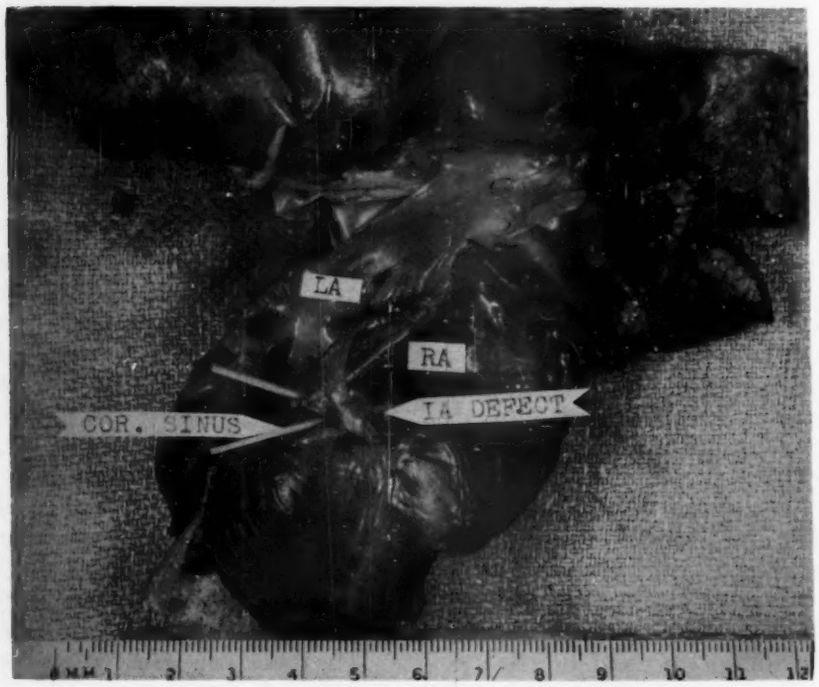

FIG. 5. Intra-atrial septal defect of the type which can be corrected.

FIG. 6. Intra-atrial septal defect and patent foramen ovale seen from the left side.

certain valvular lesions not successfully operable by other methods. These are mitral insufficiency, aortic stenosis and insufficiency. The ideal oxygenator must be satisfactory as to oxygenation of the blood, as well as simple to operate and safe.

Some of the difficulties encountered in using an extracorporeal circulation follow:

1. Technical problems: The most common problems are:

A. Anesthesia. The patient or animal must be lightly anesthetized, as in all cardiac surgery. The tracheal tube must be correctly placed and maintained in this position. The number of laboratory animals lost because of an error in this simple and primary requisite is known to all who have done this type of work.

B. The pump oxygenator. A great deal of experience is necessary before one can run any type of pump satisfactorily. This experience must be acquired by each surgical team using laboratory animals. The manufacturers of these pumps have not had the medical training to be of help in guiding the surgeon's use of the apparatus. The oxygenator system must be constantly checked, built over and changed on a trial and error basis. The rate of flow is determined by the weight of the animal. The calibration of the pump is done on the arterial end. We maintain the pump load by increasing or decreasing the flow at the venous end of the pump to keep a constant and adequate flow level. We calibrate the pump at approximately 55 to 60 ml. per minute per kg. body weight. Only if the blood pressure falls precipitously do we increase the output of the arterial end.

C. Equipment. Connections must be well made with no rough edges, and there should be no sudden changes in the size of the tubing as each variation in the caliber of the tubes increases the trauma, hemolysis and clotting factors.

D. Oxygen. The oxygen connection should be simple but adequate and the flow of the oxygen should not be too rapid. Three to 4 L. per minute has been adequate. The bubbles should be kept small by a filter.

2. Surgery: The laboratory animal must be treated and operated upon as carefully as a human being. Technical difficulties of placing the catheters correctly can be overcome only by sufficient experience. The tubes cannot be inserted too far or too short a distance. The tubing must have been perfectly cleaned and irrigated prior to, and after, sterilization. The arterial catheter should be as large as will fit into the subclavian artery (16 F.).

3. Heparinization and hemorrhage: Those

who have operated upon animals or patients in the heparin state know the difficulties of hemorrhage control. Each bleeding point must be ligated. The tubes used must be filled with heparin-saline solution to prevent clotting. Heparin must be injected into the animal first, before the tubes are placed, and the heparin must also be in the donor dog's blood. After the operation the heparin action must be counteracted by the use of protamine and the use of calcium, to be given immediately after the patient is off the pump. Since the length of the effect of protamine is always shorter than the heparin, a resurge of the heparin action may produce secondary hemorrhage. This must be watched for and corrected by testing the bleeding and coagulation times. Of utmost importance is the replacement of the blood lost by transfusion, nearly on a centimeter-for-centimeter basis.

TABLE IV
INHERENT DANGERS OF EXTRACORPOREAL CIRCULATION

1. Cardiac arrhythmia
2. Clotting or fibrination
3. Embolism { air / clot
4. Acid-base imbalance
5. Impaired metabolites
6. Impaired electrolytes

4. The donor dog: The blood must be collected with the least amount of trauma possible. We bleed the donor dogs completely, collecting the blood in 500 cc. bottles in which there is heparin and, also, in one bottle with another anticoagulant in it. The blood must be collected without trauma. The donor dog must not be oversedated or anesthetized, as these drugs will be carried to the recipient in the blood used to prime the oxygenator pump. No more than 500 cc. glucose should be used on the donor dog in order to maintain the hematocrit.

It is not my purpose to discuss the variations in the technics of different pumps and oxygenators in use elsewhere, in other cities too numerous to mention. The mechanism of the pump used may vary and the type of oxygenation may be changed but the principle of action remains the same.

The ideal pump oxygenator must do certain things: (1) It must act in the same way as the lungs and provide oxygen and remove CO_2 from the blood. (2) It must do this without interfering seriously with the clotting or the fibrination mechanism of the blood. It should not cause emboli or foaming, which cannot be removed. (3) The acid-base balance must not be changed to any great extent. (4) The metabolites and electrolytes of the body must be maintained adequately. (Table IV.)

Certain data have been collected by our experiences and are detailed as follows:

1. The preparation of the pump, particularly the removal of small bubbles from the helix and arterial end, is a tiresome and time-consuming job which must be detailed to a devoted member of the team. Most deaths are due to air emboli to the brain, so this part of the work cannot be overemphasized.

2. The pH of the donor dog's blood will fall if it is not used within one hour.

3. The dog's blood, while on the pump, goes steadily toward the acidotic side.

4. If the dog breathes too rapidly, his CO_2 component will be reduced and he will become alkalotic. Breathing above twenty times per minute causes such a state.

5. No breathing of the dog is necessary while it is on the pump. The lungs should be semi-inflated during this stage. It has been necessary for us to mouth-breathe the dog approximately every five minutes to expand the lungs and replace the contained oxygen with some nitrogen.

6. Close observation of the blood pressure is important. The pressure must be maintained pre- and postoperatively. The pressure will fall while the patient is on the pump and if the vena cava or the heart itself is manipulated. Such maneuvers should be brief. The arterial flow may have to be increased if the pressure falls. The blood pressure fall is less on arterial flow rates of 60 ml. per minute per kg. body weight than below this rate. Constant venous pressure readings prevent overloading of the right heart.

7. Meticulous hemostasis is required, especially in suturing bleeding points in the pericardium and chest wall.

8. The chest wall must be anatomically reconstructed and closed with an air-tight technic.

9. The postoperative care of the patient for twenty-four hours is most important. If skilled care is not rendered during this time,

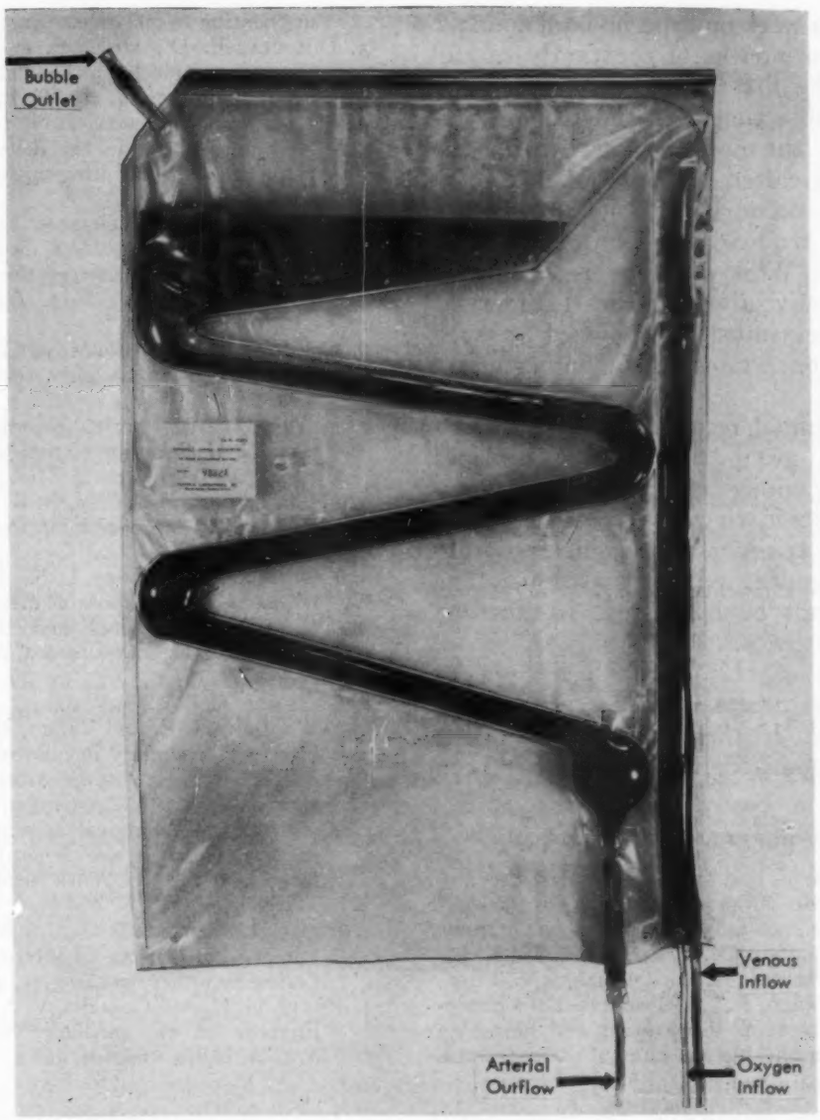

FIG. 7. Prepared oxygenator of plastic material. This apparatus reduces the technical problem of assembling and attaching the oxygenator to the pump. (Travenol Laboratories, Inc., Pharmaceutical Products Division of Baxter Laboratories, Inc., Morton Groves, Ill.) Oxygenator disposable after single use.

the patient will not survive. Fundamental and essential to surgical success are: aspiration of the chest tubes (of which there should be two), replacement of any blood loss, observation of heparin resurge, and removal of secretions and reinserting the tracheal tube if indicated.

10. The cleaning, sterilizing and the adequate connection of the apparatus with application of antifoam are all small requisites to success.

I wish to emphasize the difficulties encountered, and the time and effort one must expend to master the open heart surgery technic. Some writers tend to simplify their descriptions of this groundwork. Others have tried to by-pass some of this work and operate on human beings without this time-consuming animal experimentation. This type of surgery requires such preliminary spadework. In my opinion no clinic should be permitted to undertake open heart surgery until routine success on experimental animals can be demonstrated. (Fig. 7.)

SUMMARY

In the past year at St. Vincent's Hospital open cardiac surgery has been performed two

to three times a week on experimental animals. It required five months of constantly careful work before the first animal survived. Only after considerable time was the technic perfected so that the non-survivor became the exception. Thereafter an occasional run of failures would occur. The amount of work and teamwork required for such results is to be emphasized. While the dog is a difficult animal to keep alive after open heart surgery as described, success must be achieved on such animals before operations on human beings are attempted.

This basic animal research work should be done by each group before they consider engaging in any clinical work. Whatever pump oxygenator device is selected, it must be studied and repeatedly tried until the inherent problems have been learned by the group. The know-how cannot be by-passed. It must be learned by experience.

The purpose of this article has been to detail the efforts necessary to success in open cardiac surgery. If these points have been sufficiently emphasized, the purpose has been achieved.

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Coronary Endarterectomy

CURETTEMENT OF CORONARY ARTERIES IN DOGS

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CORONARY atherosclerosis is the cause of coronary heart disease in 95 per cent of cases. Pathologic studies have revealed that atheromatous plaques involving the first few centimeters of the coronary arteries constitute the initial lesion of coronary atherosclerosis leading to blockade of the coronary circulation and reduction of coronary flow. Although progress is being made in the understanding of medical measures aimed at prevention of coronary atherosclerosis, once this lesion is present and makes itself known by angina pectoris and/or attacks of coronary thrombosis, medical treatment is useful principally in overcoming the acute and severe phases.

Surgical approaches to the problem have been numerous and include the Thompson cardiopericardiopexy, [3], the Beck I and II procedures [12], the O'Shaughnessy cardiomentopexy [12] and other equally ingenious attempts to provide collateral circulation to the dwindling coronary channels. Experimental measures involving transplant of vessels to arterial and venous channels have not materialized into a standard procedure, and even in healthy, young, non-atheromatous experimental animals they are extremely difficult procedures attended with formidable mortality rates. The possibility of extending to the coronary arteries the operation of endarterectomy as it is performed in the descending aorta and iliac vessels was considered in a conversation with Dr. Gerson R. Biskind, to whom the author acknowledges the encouragement for the initial impetus given this work.

Since the preparation of this paper Absolon, Aust, Varco, and Lillehei [19] have published experimental work particularly indicating the feasibility of this procedure on human cadavers.

Preliminary to animal experimentation the procedures described herein were carried out in eighteen human cadavers and it was found feasible to remove the atheromatous tissue from the first portion of the coronary arteries with the curette described herein.

METHODS

A variety of methods were employed, all involving the scraping of the intima of the coronary arteries with a specially prepared hollow tubular instrument converted into a suction curette by means of a sharpened, V-shaped notch filed into its side near its tip, or with a solid tubular instrument of similar design. Experiments were carried out on twenty-three adult dogs of various sizes, ranging from 8 to 10 kg. Three approaches were employed, as follows:

1. Retrograde Coronary Curettement. The chest was opened through the left or right fifth interspace and the pericardium opened after injection of the pericardial sac with 2 cc. of 2 per cent procaine solution. The heart was delivered out of the pericardial incision, and a stitch away from the vascular bundles was placed in the apex for traction. With upward traction on the apex of the heart, two atraumatic No. 5-0 silk sutures were placed in the lower third of the anterior descending coronary artery, isolating a segment of the artery approximately 0.5 cm. long. The lower stitch was tied, closing the distal coronary artery. The upper stitch was elevated to occlude temporarily the segment of artery between the stitches. A V-shaped nick was made in the artery and a small coronary curette with the diameter of a 19 gage needle was threaded into the coronary artery and passed upward into the aorta itself. The tip of it was drawn upward into the coronary artery and the vessel itself

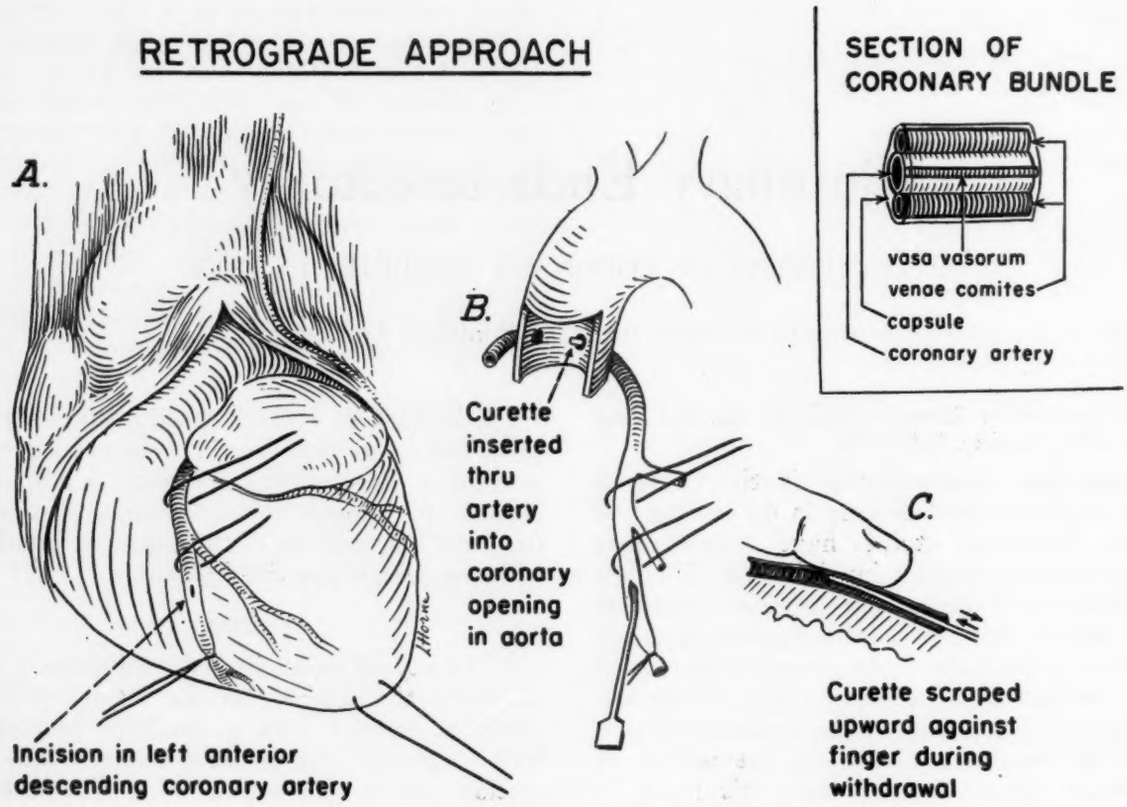

FIG. 1. The retrograde approach. Placement of sutures (A) and insertion of curette (B) into coronary artery with method of scraping (C).

briskly curetted, the V-shaped notch in the curette drawn against the index finger of the opposite hand on the outside of the heart. After withdrawal of the curette the second suture was tied, closing the coronary artery in this area. Occasionally, a third stitch was placed as a figure-of-eight suture over the actual incision into the coronary artery to control oozing from the collateral vessels in this segment. The chest was closed in the usual manner. (Fig. 1.)

Six dogs were operated upon in this manner, as follows:

Dog 197, operated upon on November 8, 1955, and sacrificed on February 8, 1956: Sections were taken from above and below the site of ligature and also from the right coronary artery for comparison. The sections showed mild inflammatory reaction in the adjacent loose tissues, and patent coronary vessel in the sections above and below the ligature. The sections of the right coronary artery contrasted in the absence of the tissue reaction about the vessel.

Dog 212, operated upon on January 30, 1956, and sacrificed on February 8, 1956: At the time of sacrifice sections were taken from below and above the site of ligature. The section from above the ligature showed an acute inflammatory process in the adjacent loose tissues. The artery itself was patent but showed subendothelial and endothelial thickening. The section from below the ligature showed patency of the vessel with adjacent inflammatory reaction.

Dog 226, operated upon on March 8, 1956, and sacrificed on May 9, 1956: Sections were taken from below and above the level of ligature and along the course of the coronary artery up to the aorta. In all sections taken the vessels were patent and no microscopic changes were found.

Dog 229, operated upon on April 16, 1956, and sacrificed on May 9, 1956: Sections were taken from the site of ligature, above the ligature and along the course of the coronary artery up to the aorta. The vessel above the ligature and along the course of the coronary artery was patent and showed no microscopic changes, but the section from the site of ligature showed the vessel to be thrombotic.

Dog 233, operated upon on April 27, 1956, and sacrificed on May 9, 1956: Sections were taken from below and above the ligature on the anterior descending coronary artery, and both the distal portion of the vessel as well as the vessel proximal to the ligature were found to be completely patent. Microscopic sections showed mild tissue reaction surrounding the vessel but the vessel to be completely patent above and below the ligature. In the section between the ligature, the vessel was thrombotic.

Dog 221, operated upon on February 7, 1956, and died the same day: The heart was removed and sections were taken from the left coronary artery at the following sites: just above the ligature, a cross section from the circumflex and a cross section from high on the left anterior descending coronary artery. All sections showed patent coronary artery and some ecchymosis in the adjacent tissues in the lower section only.

2. *Transaortic Approach.* The chest was opened from either the left or right third interspace. The aorta was dissected free from the surrounding tissues and a nylon tape was placed around the aorta and tied securely over a glass rod, which was immediately removed so that the diameter of the aorta was reduced approximately 30 per cent. The curette was fashioned from a long spinal puncture needle, the stylet of which previously had been removed and the end of the needle filed off to a dull, rounded edge. Approximately 0.5 cm. from the end a V-shaped notch, sharpened to a "razor edge" on its flat portion, was made in the side of the needle using a triangular file. The stylet was reinserted and sharpened to a fine point in order to puncture the aorta. With the stylet in the modified needle the aorta was punctured through the nylon tape and the stylet then removed, with the now blunt-tipped instrument within the aorta. The coronary orifices were found by palpation with the index finger and the instrument was threaded into a coronary orifice and first portion of the corresponding coronary artery. The coronary vessels were briskly curetted with the sharpened edge of the instrument. The instrument was then withdrawn and the nylon tape drawn to the side of the puncture opening in the aorta. With a suction tip over the spurting opening in the aorta a figure-of-eight No. 5-0 atraumatic silk suture was placed to close the puncture point in the aorta. The pericardium was closed with interrupted silk sutures and the chest was closed after inflating the lung. (Fig. 2.)

Four dogs were operated upon in this manner, as follows:

Dog 141, operated upon on October 6, 1954, and sacrificed on August 3, 1955, having undergone in all, all three methods: At the time of sacrifice Schlesinger's injection of the coronary arterial tree was prepared, showing patent coronaries throughout with the exception of the tied-off segment of the left anterior descending coronary artery.

Dog 138, operated upon on October 13, 1954, and died on the table from exsanguinating hemorrhage: Autopsy of the heart revealed a wide split in the right lateral aspect of the aorta and extending to the aortic ring. The left anterior descending branch of the coronary artery was uninjured.

Dog 139, operated upon on October 20, 1954, and died on the table from exsanguinating hemorrhage from the side of the aorta when we attempted to suture the aorta: Autopsy revealed the aorta to be split by the clamp just above the aortic valves. The coronary artery which was entered by the curette was patent throughout and showed no sign of rupture.

Dog 157, operated upon on January 26, 1955, and sacrificed on July 27, 1955: The heart was removed and injected with Schlesinger's solution, and skiagrams of the injected heart were taken. These skiagrams showed excellent injection of the coronary trees, with no evidence of occlusion due to the scraping.

3. *Combined Carotid and Left Chest Approach.* An incision was made over the right carotid artery and the artery was isolated. Two strands of silk were placed around it and a V-shaped notch was made in the segment between the strands. Through the V-shaped incision a long tubular curette, similar to the aortic curette but without a stylet, was inserted down the carotid artery and threaded to the aortic valve, which was located by the clicking sensation imparted through the curette. The left chest was then opened through the third interspace and the pericardium was opened after injection with 2 cc. of 2 per cent procaine solution. The curette was then threaded into the left coronary artery by palpating the origin of this vessel in the posterior left aspect of the aorta and following the finger with the tip of the curette. It was not found possible to

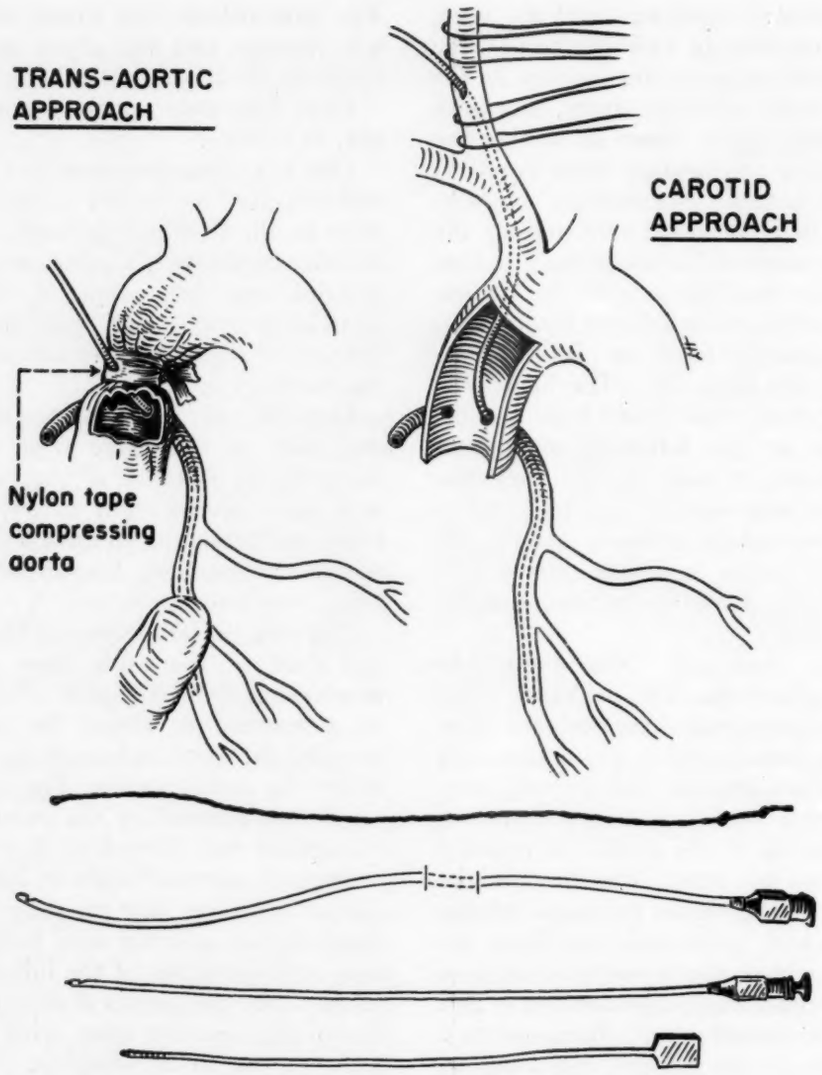

FIG. 2. The transaortic and carotid approaches. Methods of entering coronary arteries from above and types of curettes used.

enter the coronary artery blindly without opening the chest, the usual result being rupture of the coronary artery or insertion of the instrument through the wall of the heart. With the open chest, however, no difficulty was encountered in threading the curette into the coronary artery, and by holding the curette against the index finger it was possible to curette the intima of the first few centimeters of the vessel. In most cases it was found possible to thread the curette from the carotid artery down to within 3 cm. of the tip of the ventricle into the anterior descending coronary artery. After withdrawal of the curette, the opening in the carotid artery was closed with interrupted No. 5-0 silk sutures without sacrificing this vessel. The pericardium and chest were closed in the usual manner. (Fig. 2.)

Four dogs were operated upon in this manner, as follows:

Dog 142, operated upon on October 27, 1954, and sacrificed on November 26, 1954: Examination revealed patency of the left anterior descending coronary artery with no evidence of thrombosis but an inflammatory and fibrotic reaction surrounding the artery.

Dog 148, operated upon on December 1, 1954 and died on the table: The curette was passed into the left circumflex coronary artery, which ruptured. Sutures were placed in the

ruptured portion but the heart went into ventricular fibrillation and it was impossible to defibrillate the heart.

Dog 164, operated upon on April 21, 1955, and sacrificed on August 15, 1955: The right carotid artery was exposed and the long curette was passed into the left anterior descending coronary artery blindly. The coronary artery was injected with diodrast® and an immediate skiagram was taken which faintly visualized the vessel. The instrument was withdrawn and the carotid vessel sutured. At the time of sacrifice on August 15th Schlesinger's injection of the coronary revealed no areas of thrombosis.

Dog 165: This animal was operated upon on May 15, 1955, and died on the table due to rupture of the coronary artery.

CONCLUSION

Instrumental exploration of the coronary arterial tree, both through a transaortic approach and by an approach through the terminal portion of the coronary arteries, was successfully accomplished in dogs. The retrograde method was found the most practical and quickest, and involved minimal mortality.

SUMMARY

Experiments have been performed which showed that in the living dog's heart it is possible to pass instruments in and out of the coronary arteries, either from above or below. Injuries to the intima of the coronary arteries may be produced without causing death or disability of the animals. Technics and approaches, as well as factors and management of the procedures, have been described.

Acknowledgments: Mr. Jesse Linzer for making the curette; Drs. Felix Pearl and Meyer Friedman for many suggestions and technical advice during the course of the experiments; Dr. David Linder for kind cooperation in cutting and preparing microscopic sections; Mrs. Beryl Kallemeyn for technical assistance; Mrs. Louise Horne for illustrations.

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Partial Gastric Resection for Duodenal or Marginal Ulcer

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THE recent surgical literature contains many excellent reviews of the surgical management of peptic ulcer. These reviews discuss various procedures such as vagotomy-gastrojejunostomy, radical partial gastrectomy, the Billroth I gastric resection, and vagotomy and hemigastrectomy. In the light of the enthusiasm for these last procedures exhibited by their proponents, it has seemed worth while to review our own series of ulcer cases treated by the technic of radical partial gastrectomy with modified Billroth II anastomosis.

TABLE I
INDICATIONS FOR OPERATION

Indications	No. of Cases	Per cent	
Acute bleeding	25	12.5	40
Elective operation for bleeding	26	13.0	
Obstruction	29	14.5	
Intractability	34	17.0	60
Intractability plus previous perforation	33	16.5	
Intractability plus previous hemorrhage	34	17.0	
Intractability plus previous hemorrhage, plus previous perforation	19	9.5	

From the opening of the Cleveland Veterans Administration Hospital in July, 1946 until February, 1955, 366 partial gastric resections were performed for all causes. From July, 1946 to February, 1954, 200 such operations were performed for duodenal or marginal ulcer and constitute the basis for this report. It is the purpose of this paper to report our experience with these cases, dwelling particularly on their immediate pre- and postoperative course and also including an estimation of the result obtained by surgery. In these 200 cases, 222 ulcers or ulcer scars were found. Follow-up studies, obtained on 195 cases, have varied from three months to five years; a majority of patients were followed up two to three years.

INDICATION FOR OPERATION

As can be seen in Table I, 25.5 per cent of these operations were performed for bleeding, the operations being either of emergency or elective nature. In general, patients bleeding acutely were admitted to the Medical Service, treated medically, were seen and followed up by the Surgical Service, and were operated upon only after medical and surgical consultants had decided the patient would most likely bleed to death without operation. If the bleeding stopped on medical management, elective resection was performed about three weeks later on those patients who had bled two or more times—bled enough, that is, to require medical care. In the older patient age group, other things being equal, there was a strong inclination on the part of the surgical staff to urge elective operation after a single massive hemorrhage.

In all patients with pyloric obstruction an attempt at medical management was made, usually for seven to ten days, before they were transferred to surgery. It was of interest to us, as has been pointed out by others, that a considerable number of these patients were obstructed not by cicatricial stenosis but rather by edema and presumably pylorospasm due to an acute ulcer.

The largest group (120 or 60 per cent of the total) was composed of the intractable cases, that is, patients who for one reason or another

were refractory to medical management. No patient in this group was operated upon without at least one good trial of medical management, either in our hospital or under medical care known by us as good. Many patients had repeated Medical Service hospitalizations. Almost all these patients either were admitted first to the Medical Service or before being taken to the operating room were seen by a gastroenterologic consultant who concurred in the opinion that operation was indicated. Of this group, less than one-third had had no previous complications. The remaining two-thirds had had either perforation or hemorrhage, or both, and in many cases multiple perforations or hemorrhages.

VITAL STATISTICS

All patients were men but one. The one female patient was operated upon for stomal ulcer following gastrojejunostomy elsewhere for duodenal ulcer. The average age was forty-five, the oldest patient being eighty-two and the youngest twenty-four. The percentage of patients between thirty and fifty was 61.5. Eighty-eight and one-half per cent of the patients were married or had been married. As for occupation, most patients were laborers, clerks, millworkers, service station attendants, mechanics and the like. Few patients had completed high school and only one or two of the entire group was a college graduate.

ROENTGENOGRAPHIC STUDIES AND SURGICAL FINDINGS

Of the entire group, 193 patients had gastrointestinal roentgenograms taken preoperatively. The seven who did not were operated upon for acute bleeding without benefit of x-ray examination. At present we believe that in most, if not all, acute bleeders who have never undergone a previous gastrointestinal series, an emergency x-ray examination of the esophagus, stomach and duodenum should be performed if urgent surgery is being seriously contemplated. This is of particular importance if there is a history or findings suggestive of cirrhosis or if for any reason there is any doubt as to the diagnosis of ulcer. Such an examination may or may not help, but in good hands we have not seen it do harm and in some cases it may save the patient a hazardous laparotomy. Of the 193 patients who had gastrointestinal x-ray examinations, there was pyloric obstruction in ten to such a degree that the x-ray diagnosis simply was pyloric obstruction. A deformed duodenal bulb was found in 183 patients, fifty-one of whom also had a demonstrable ulcer crater. Seven marginal ulcers are included in this group and fourteen gastric ulcers were diagnosed. (Table II.)

TABLE II
ROENTGENOGRAPHIC STUDIES

Findings	No. of Cases	Per cent
Duodenal crater	51	25.5
Duodenal deformity	183	91.5
Pyloric obstruction	10	5
No examination performed	7	3.5
Marginal ulcer	7	3.5
Gastric ulcer (crater)	14	7.0

At operation or according to the pathologic report, all patients had a duodenal crater or scar. In addition, there were fifteen gastric ulcers or scars and seven marginal ulcers. Four pyloric ulcers were reported by the pathologist and for want of a better place to put them, they are included in this study as duodenal ulcers.

SURGICAL TECHNIC

Some 70 to 80 per cent of these procedures were performed by members of the surgical resident staff. The abdomen was opened usually through a right or left rectus muscle–retracting incision, and a two-thirds to four-fifths (estimated) resection was performed. A considerable effort was made to remove all antral mucosa, preferably by cutting across the duodenum, although in two patients antral exclusion procedures with excision of antral mucosa were performed; in one of these patients a marginal ulcer developed in about eighteen months. The ulcer or ulcer scar was removed in 110 cases, was left in in ninety-four cases, and in eighteen cases it could not be determined from the record whether or not the ulcer was removed. In many of the patients in whom the ulcer was left, it was believed that prolonged or vigorous attempts at removing it might result in injury to the common bile duct. Only in patients undergoing operation for acute bleeding do we believe that considerable effort should be made to remove the ulcer, for in this series there were two patients operated upon

for bleeding in whom the ulcer was left who bled to death postoperatively, presumably from the ulcer.

All anastomoses were Billroth II modifications as indicated in Table III; in all except one or two the afferent jejunal loop was anastomosed to the lesser curvature of the stomach. The type of anastomosis appeared to make little difference in the late postoperative course. Relatively early, however, there were two serious complications among the long loop anterior group. One patient had an acute volvulus of the afferent limb with complete duodenal obstruction and serious compromise of the circulation in the twisted loop. This occurred about two weeks after operation, just as the patient was ready for discharge, and necessitated a second operation during and following which the patient almost lost his life. The second complication occurred in a patient who had undergone an anterior Polya anastomosis; the afferent limb was twisted so as to obstruct the efferent limb, necessitating a second laparotomy about three months after gastric resection. In one of the patients with a posterior anastomosis, the stoma failed to open. Reoperation revealed that the anastomosis had pulled up through the mesocolon, completely obstructing the efferent limb.

TABLE III
TYPE OF ANASTOMOSIS

Type of Anastomosis Performed	No. of Cases	Per cent
Anterior Polya	48	24
Posterior Polya	4	2
Anterior Hofmeister	15	7.5
Posterior Hofmeister	133	66.5

EFFECT OF OPERATION ON WEIGHT

Of the entire group of 200 patients, 102 were weighed shortly before operation and at follow-up three months or more later. Of this group, 61.4 per cent lost weight and 36.6 per cent gained weight, while in 2 per cent there was no change in weight. Of the patients who gained or lost more than 5 pounds, 70.6 per cent lost weight while 29.4 per cent gained weight. This is in fair agreement with the figures of Allen and Welch [1] who noted a weight loss in 61 per cent of their patients, a weight gain in 23 per cent and no change in 16 per cent. In many cases, however, an admission weight is not a fair indication of the weight in good health of these patients, but rather is probably less than the average weight in good health. Therefore, the number of patients who lost weight following operation, when their average weight in good health is considered, is probably greater than these figures indicate.

TABLE IV
POSTOPERATIVE COMPLICATIONS

Complications	No. of Cases	Per cent
Pulmonary	38	19
Leaking duodenal stump	5	2.5
Wound dehiscence	4	2.0
Major wound infection	2	1.0
Obstruction of gastroenterostomy stoma	10	5.0
Pulmonary infarction	1	.5
Thrombophlebitis	11	5.5
Incisional hernia	1	.5
Jejunal fistula	1	.5
Twisted anterior loop	2	1
Injury to common bile duct	1	.5
Cerebral thrombosis	2	1.0
Postoperative hemorrhage	4	2.0
Coronary infarction	1	.5
Totals	83	41.5

POSTOPERATIVE COMPLICATIONS

In Table IV a list of fatal and non-fatal postoperative complications is given. Altogether, eighty-three patients or 41.5 per cent of the entire group had some sort of postoperative complication, a somewhat higher figure than that given by Metcalf et al. [25]. Of the five patients with leaking duodenal stumps, three were treated conservatively (gastric suction and antibiotics) and recovered. A fourth patient had a subhepatic abscess drained and recovered, and the fifth recovered after multiple operative procedures and prolonged hospitalization. The jejunal fistula occurred in this same patient, it is believed, due to pressure necrosis caused by an intra-abdominal Foley catheter passed down toward the leaking stump. This fistula was surgically closed. Ten patients had obstruction of the gastrojejunostomy stoma which responded to conservative management in most cases; only one patient

required re-exploration. In two of the four patients with severe postoperative hemorrhage, the bleeding was believed to be from an ulcer left in place; both patients exsanguinated. Two other patients were re-explored, the anastomosis taken down and the suture line oversewn with successful control of bleeding from it. Several other patients had more blood in their suction bottles than usual but did not require operation and stopped bleeding spontaneously.

RESULTS

Our estimate of the results obtained by operation is listed in Table v. Results were considered excellent in those patients who gained weight, maintained their weight or lost less than 5 pounds, worked regularly, had no pain and had few to no food idiosyncrasies. Good results are those in which all these criteria were not fulfilled: the patient did not or could not work all the time because of symptoms relating to his gastrectomy; he maintained a moderate to considerable weight loss; he complained of excessive weakness and fatigue; he had to limit his diet moderately; or he had a moderately severe dumping syndrome. This group, composed of 60.5 per cent of all patients, is the largest and appears to correspond fairly well with the combined good and fair result cases (65.6 per cent) of Metcalf et al. [25], as do our excellent results (27.5 per cent) with their excellent results (22.7 per cent).

Thirteen cases were considered failures; five were due to marginal ulceration which in every instance occurred less than two years postgastrectomy, and eight were due to "vague symptoms." Three of the patients with marginal ulcers were admitted because of gastrointestinal bleeding, one because of perforation and one because of moderately severe pain. All these patients to date have been cured by vagotomy except one who refused operation. The term "vague symptoms" just mentioned applies to a group of eight patients who made frequent return visits to the hospital complaining of all sorts of things: weakness, fatigue, severe dumping, excessive weight loss, inability to work, vague pain, nausea and vomiting, and so on. These patients were so severely disturbed by such symptoms as to be considered gastrectomy failures.

There were six deaths. Five of these occurred in patients operated upon for acute bleeding, making our mortality rate in operating upon acute bleeders 20 per cent (twenty-five operations, five deaths). Two of these five patients bled to death postoperatively with ulcers left *in situ*, one died of cardiac arrest in the recovery room about ten minutes postoperatively (this patient had congenital heart disease and pulmonary tuberculosis), one died of pulmonary infarction and one of coronary infarction. The one death in an elective gastrectomy was believed due to a cerebrovascular accident. The over-all mortality rate therefore was 3.0 per cent. The total of good and excellent results was 88 per cent, failures, deaths and no follow-up 12 per cent.

TABLE V
RESULTS

Results	No. of Cases	Per cent	
Excellent	55	27.5	88
Good	121	60.5	
Failure	13	6.5	12
Death	6	3.0	
No follow-up	5	2.5	

COMMENTS

A majority of these patients have been seen in follow-up by one of the authors, and although most patients were seen repeatedly by various physicians, usually one of us saw a single patient only once. The type of operation performed is well standardized and has withstood the test of time and many thousands of performances. In our hands it was strikingly successful in relieving the patient of the ulcer pain, obstruction or bleeding if that happened to be the primary indication for operation, and from this point of view it is an excellent and worth while procedure almost without qualification. However, a number of disagreeable side effects of the operation have been observed. Weight loss and an inability to gain weight postoperatively have been observed in a large number of patients. Excessive weakness and fatigue far above that noted preoperatively is frequently present and appears to be directly related to weight loss in most cases. The incidence of a dumping syndrome that has been really incapacitating has been low, but the incidence of food idiosyncrasy has been quite high. A majority of patients on close question-

ing will state that certain foods (notoriously milk shakes and sweets including desserts) cause nausea, sweating, weakness and sometimes vomiting. Only 27.5 per cent of our patients have been free from these side effects.

It appears, therefore, that radical partial gastrectomy according to the Billroth II technic may not be the final answer to the problem of the surgical management of duodenal ulcer. Of the recently popular procedures, hemigastrectomy and vaogotomy seems to have promise and we are presently trying them out, but thus far have too few cases to comment on. It is to be hoped, at any rate, that this or some other procedure can be shown to be better and its value firmly established.

SUMMARY

1. It is important to remove a duodenal ulcer only in patients operated upon for acute bleeding.

2. Weight loss is an important, measurable and frequent sequela of operation.

3. Easy fatiguability and weakness are frequent undesirable sequelae.

4. Radical partial gastric resection relieves ulcer symptoms in most cases and, for that reason, it is probably the best single extensively used operation for duodenal ulcer. Because of undesirable side effects it is hoped a better procedure can be devised and its value solidly proved.

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Choledochal Cyst

WITH A REPORT OF AN INTRADUODENAL CHOLEDOCHAL CYST

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CHOLEDOCHAL cyst is an idiopathic dilatation of the common bile duct, probably congenital in origin. They are of interest and should be reported because of their rarity and because the ultimate prognosis seems to bear a direct relationship with the surgeon's ability to recognize the pathologic condition with which he is dealing.

Vater described the first case of choledochal cyst in 1723 [*13*]. Todd reported a "remarkable enlargement of the biliary duct" in 1818 [*5*]. Approximately 200 cases have been reported in the literature [*16*]. Shallow et al. [*14*] in 1943 presented an excellent review of the literature in which they collected 175 cases, 25 per cent of which were reported by Japanese authors. The condition has also been referred to as congenital cystic dilatation of the common bile duct, idiopathic cystic dilatation of the common bile duct, choledochocele, diverticulum of the choledochus, choledochus cyst and megalocholedochus.

PATHOLOGY

Choledochal cyst is not a true cyst of the common bile duct, but is a localized dilatation or enlargement of the duct. Characteristically, this dilatation is limited to the supraduodenal portion of the duct and is circumferential. The dilatation may involve only the common duct or it may also involve the common hepatic duct so that the cystic and hepatic ducts enter the cyst as separate ducts, separated by cyst wall. The dilatation of the common duct as seen in choledochal cyst is, however, usually characterized by the fact that the dilatation is localized with no evidence of dilatation of the gallbladder, common hepatic or hepatic ducts and no dilatation of the common duct distal to the site of the choledochal cyst. These findings are very consistent in the reported cases and tend to help differentiate this lesion from the usual case of obstruction of the biliary tract which is characterized by a diffuse, even dilatation of the entire proximal biliary tree, including the gallbladder, such as is seen in carcinoma of the ampulla. The cysts vary greatly in size, having been reported as containing from 30 cc. to several thousand cubic centimeters of fluid. Shallow et al. [*15*] reported a case in which the cyst contained 5,800 cc. of bile-stained fluid. Keeley [*11*], in 1948, reported a case in which the cyst contained six smooth, yellowish white stones and stated that only three other cases of cysts containing stones had previously been reported. Shallow et al. [*14*] in a review of the literature, found only two instances in which the cyst had extended far enough retroduodenally so that the pancreatic duct opened directly into the cyst. The gallbladder is usually not dilated and is frequently adherent to the wall of the cyst. The cyst wall itself is usually thick, measuring 4 to 5 mm., and apparently tough enough to cause no difficulty in suturing. Microscopically, the cyst wall is characterized by fibrous tissue and the mucosa of the cyst wall has usually been denuded. Occasionally remnants of mucosa remain, and sometimes smooth muscle fibers and elastic tissue are still identified within the cyst wall. Irwin [*10*] reported an unusual case of a thirty year old patient with a choledochal cyst which contained carcinoma. He postulated that this represented cancerous change from chronic irritation in a long-standing cyst. The fluid contained within the cyst is characteristically described as bile-stained, although in occasional cases acute suppuration

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has taken place and the fluid has been described as purulent. The liver is frequently enlarged and cirrhosis is common.

ETIOLOGY

Many theories have been advanced regarding the etiology of choledochal cysts but none of them adequately explain all cases, with the exception of the theory that the cyst is of congenital origin. Obstruction due to stenosis, stricture or kinking has been found distal to the cystic dilatation in many cases, but this finding does not explain the localized dilatation of the common duct, unless a congenital weakness is present at the site of dilatation. Many cases of cystic dilatation of the common duct have been described in which careful exploration has failed to reveal any evidence of obstruction distal to the cyst. The most tenable explanation of the etiology is that the cyst is a result of a congenital weakness of the duct wall which can dilate under normal intraductal pressures, but which may be associated in some cases with partial obstruction due to stenosis secondary to cholangitis or another abnormality. Kinking of the duct distal to the cyst probably is the result of torsion or pulling on the common duct as a result of the weight of the cyst itself, therefore explaining the characteristic intermittent symptoms frequently reported with choledochal cysts, the cyst emptying or filling according to the position it assumes as the pressure increases and decreases in relationship to the position of the distal common bile duct.

DIAGNOSIS

Needless to say, the preoperative diagnosis of choledochal cyst will never be made if it is not considered in the preoperative differential diagnosis. Unfortunately, due to its rarity, it is not considered preoperatively and the diagnosis is frequently missed at the operating table, usually with disastrous results. Shallow et al. [*14*], in a review of 175 cases collected from the literature, found that 77 per cent of the patients were female and 76 per cent of the patients were twenty-five years of age or younger. The preoperative diagnosis was correctly made in only 8.6 per cent of the cases and mentioned as a possibility in 4 per cent. In correlation, they found that with the correct preoperative diagnosis the mortality rate was 36 per cent, while with an incorrect preoperative diagnosis the mortality rate was 62 per cent. In their review they found that the mortality rate in those patients in whom the true pathologic condition was not recognized even at surgery was 83 per cent, thus emphasizing the necessity for a correct operative diagnosis and, better still, a correct preoperative diagnosis.

The triad of abdominal pain, tumor and jaundice is described frequently enough to make it the outstanding feature of this disorder. Shallow et al. [*14*] found tumor present in 77 per cent of the patients, jaundice in 70 per cent and pain in 59 per cent. It is obvious from the percentages that all three do not occur in every case, but one or two of the triad is almost always present in a given case. Pain is usually complained of in the right upper quadrant of the abdomen and may be indistinguishable from that frequently complained of in chronic cholecystitis with acute exacerbations. It is rarely described as being as severe as that of acute cholecystitis or renal colic. The abdominal tumor, if present, may vary greatly in size and apparent consistency, having been described as a well defined, small, circumscribed mass just below the liver edge to a large cystic resistance palpable throughout the entire epigastrium and right side of the abdomen extending down to the iliac crest. When present, jaundice is of the obstructive type and is confirmed by the usual blood, urine and stool examinations. The van den Bergh test is predominantly positive in the direct reaction. Fever is not a prominent symptom and when present is usually indicative of cholangitis with infected cyst fluid or evidence microscopically of acute inflammation of the cyst wall. The patients may complain of fatty food intolerance, characterized by a postprandial feeling of fulness and flatulence rather than any acute episodes of colicky pain or vomiting. There is very wide variation in the age; Gross [*9*] mentions a case discovered in a fetus, and Shallow et al. [*14*] report a case in a fifty-eight year old white man. Dickinson et al. [*6*] reported the case of a two-month old white female infant who had had jaundice since three days of age. There was no mass palpable preoperatively and a preoperative diagnosis of biliary atresia was made. A typical choledochal cyst was found and treated by primary anastomosis; the postoperative course was benign. In spite of adequate preoperative

diagnosistic studies, the diagnosis could not be suspected. An example of this is a case reported by Goldman et al. [7] of a thirty-seven year old woman with pain in the right upper quadrant, flatulence and a history of painless jaundice twelve years prior to admission with no recurrence of the jaundice. No mass was palpable on admission and a gastrointestinal series was reported as within normal limits. At exploratory laparotomy, a choledochal cyst described as being 7 cm. in diameter was found.

One of the most striking features of choledochal cyst is the chronicity and intermittency of the clinical course. It has been emphasized in several reports that the patients gave a history of enlargement of the abdomen following meals, before the occurrence of pain, jaundice or other symptoms made hospitalization necessary. It is not uncommon in these cases to find that palpation of the abdominal tumor reveals a variation in size and consistency at different times, apparently dependent upon the amount of fluid contained within the cyst at the time of examination. Not infrequently patients will complain of intermittent pain in the right upper quadrant over a number of years. They also give a prolonged history of intermittent attacks of jaundice. This history of chronicity and intermittency is very helpful in ruling out neoplastic diseases of the abdomen in those patients with a very tense, solid-feeling cyst. Illustrative of the chronicity and intermittency of these cases is a case report by Keeley [11] of a thirty year old white man who had had gastric disturbances in infancy with intermittent symptoms of pain in the right upper quadrant followed by jaundice throughout his life. Between the ages of twenty-three and thirty the attacks became more frequent. At the age of thirty exploration was performed, revealing a choledochal cyst which was treated with a Roux-Y anastomosis with a successful result. Kjellberg [12] and Lumpkin et al. [13] have both reported one case in which they aspirated the choledochal cyst with a needle and inserted radiopaque material as a diagnostic procedure. They both concluded that there were no ill effects from this procedure and that it was an excellent diagnostic adjunct, allowing them to make a correct preoperative diagnosis in their respective patients. Unusual cases have been reported which would defy preoperative diagnosis. For example, Blegen et al. [2] report a case of spontaneous perforation of a choledochal cyst with abscess formation, and Blocker et al. [3] report the case of a traumatic rupture of a choledochal cyst which terminated fatally. The latter patient was a fourteen year old boy who had been completely asymptomatic prior to trauma. At autopsy no evidence of stricture or obstruction of the distal common bile duct was found although the patient had a choledochal cyst measuring 7 by 5 to 7 cm. These findings led the authors to conclude that this case added more weight to the theory that choledochal cyst is a congenital defect rather than secondary to obstruction.

LABORATORY STUDIES

The usual laboratory studies may be entirely within normal limits, but in those patients with jaundice the typical reactions will be indicative of obstructive jaundice with a positive direct reaction to the van den Bergh test, bile in the urine, negative urine urobilinogen and negative urobilinogen in the stool. These laboratory tests will typically fluctuate, and stool and urine specimens will frequently show varying results if repeated often enough. Liver function studies may or may not be abnormal and an elevated sedimentation rate is indicative of cholangitis.

X-RAY STUDIES

Oral cholecystograms typically reveal no visualization.

Intravenous cholangiograms have not been mentioned in the literature but should be very helpful in diagnosing choledochal cyst in spite of the fact that visualization is not obtained in a large percentage of patients with jaundice. In these patients in whom visualization of the choledochus is obtained with an intravenous cholangiogram, the preoperative diagnosis would be assured.

Aspiration of the cyst and diagnostic injection of water-soluble radiopaque material has been reported by two authors, as previously stated. It was their belief that there were no ill effects from this instillation of dye and a preoperative diagnosis was definitely made in both cases. This method of examination probably warrants further cautious evaluation.

Intravenous pyelograms should be taken in the presence of an undiagnosed tumor in the right upper quadrant of the abdomen and will, in most instances, rule out tumors of the right kidney and massive hydronephrosis of the right

kidney. If any question remains following this study, a retrograde urogram with retroperitoneal air or oxygen insufflation may be taken and should definitely rule out the kidney as the site of the abdominal mass. If the choledochal cyst is large enough to cause appreciable pressure on the right ureter, slight hydronephrosis or delay in emptying of the right kidney may be noticed.

Flat films of the abdomen, an upper gastrointestinal series and barium enemas should be very helpful in the differential diagnosis of a mass in the right upper quadrant if the mass is large enough to cause the typical displacement seen with a choledochal cyst. In the typical case of choledochal cyst, evidence of extrinsic pressure on the gastrointestinal tract will be seen with no intrinsic defects unless they are purely coincidental. The typical displacement will reveal that the pylorus and first portion of the duodenum is displaced anteriorly, downward and to the left with the splenic flexure of the colon and the transverse colon displaced anteriorly and inferiorly. The history, physical examination and x-rays showing typical displacement of the duodenum tend to differentiate choledochal cyst from tumors of the kidney, hydronephrosis of the right kidney, omphalomesenteric cyst, omental cyst and duplications of the duodenum which characteristically occur on the lesser curvature of the duodenum and do not tend to attain the size of the usual choledochal cyst.

TREATMENT

Successive treatment depends upon either a preoperative diagnosis or a correct operative diagnosis while the patient still remains in an operable condition. Some of these cases have been reported with a fatal outcome because the condition was not recognized at the operating table and was treated with an unphysiologic procedure, or was not recognized until so much dissection had been accomplished that the patient was in surgical shock before definitive treatment could be instituted. Shallow et al. [*14*], in their review of 175 cases, found the following: of forty patients treated with external drainage, 83 per cent died; of twenty-four treated with external drainage with secondary operation consisting of a biliary intestinal anastomosis 29 per cent died; of sixty patients who had primary anastomosis of the biliary system to the gastrointestinal tract 27 per cent died. In evaluating twenty-four miscellaneous procedures in which the true pathologic condition was not recognized at surgery, the mortality rate was 83 per cent.

Those patients who receive no treatment will eventually die of biliary obstruction or complications. Treatment by repeated aspiration of the cyst is obviously inadequate as the basic pathologic condition would be unaltered and the fluid will reaccumulate. Injection of sclerosing agents is obviously unwise as it would fail to correct the condition and might be fatal. As shown previously, the mortality rate with external drainage is so high that it obviously condemns this method of treatment. External drainage with secondary anastomosis of the biliary tract to the gastrointestinal tract may be necessary in some poor risk patients, but as a routine measure it is to be condemned as it necessitates two operations and makes the second and definitive procedure much more difficult technically.

Dennison [*5*] reports a large choledochal cyst filled with 4,000 cc. of fluid which he stated was infected and much too large to excise or anastomose to the gastrointestinal tract. He therefore excised as much of the cyst wall as possible and treated the remaining wall with pure carbolic acid. He states that the postoperative course was benign and the patient was perfectly well at the time of five-month follow-up. This method of treatment is mentioned only to be condemned because it is obvious that, in the usual case of symmetric circumferential dilatation of the common bile duct, excision of a portion of the cyst wall with treatment of the remaining portion with pure carbolic acid would probably end fatally since no provision for continuity of the biliary system is provided. Excision of the mass with establishment of direct continuity between the biliary system and the gastrointestinal tract with a choledochoduodenostomy or a choledochojejunostomy built on the Roux-Y principle, would seem physiologically to be the treatment of choice in choledochal cysts. Shallow et al. [*14*] reported a case in which the cyst was extirpated and the hepatic ducts were anastomosed to the duodenum with a successful outcome. The advantage of this method of treatment over primary anastomosis of the cyst to the gastrointestinal tract is that there would be no remnants of the cyst left behind that theoretically would allow bile to stagnate,

become infected and thus lead to further difficulty. Although primary extirpation of the choledochal cyst would appear physiologically to be the most advantageous treatment, it is believed that in the majority of cases this method is to be condemned in that it entails too radical and too dangerous a dissection to treat a condition which can be more easily and satisfactorily treated by a technically simple anastomosis of the cyst itself to the gastrointestinal tract with a minimum of untoward effects.

Primary anastomosis between the cyst and the intestinal tract is without doubt the treatment of choice in choledochal cyst. If drainage is instituted with an adequate anastomosis, the cyst will shrink and postoperative roentgenograms will reveal a return to normal anatomic position of the stomach, duodenum and colon. It is also believed that if an adequate anastomosis is made, ascending cholangitis will not be a problem. Walters [*17*] reviewed 186 cases of stricture of the common and hepatic bile ducts and concluded that following choledochoduodenostomy, cholangitis is not secondary to reflux of gastrointestinal contents but is secondary to obstruction at the site of anastomosis. There are without doubt exceptions to this concept, but if in the majority of instances an adequate anastomosis between the cyst and the duodenum will prevent cholangitis, it certainly makes this simple technical procedure the treatment of choice when compared to the more complicated Roux-Y type of anastomosis between a limb of defunctionalized jejunum and the cyst proper. Shocket et al. [*16*] reported the successful use of a Roux-Y type of anastomosis to a choledochal cyst in a patient, followed by a benign postoperative course. They state that prior to their report only four other choledochal cysts treated with a Roux-Y type of anastomosis had been reported, and that only one of the previously reported patients had had a stormy postoperative course. Keeley [*11*] performed a Roux-Y anastomosis with a 12 inch limb of defunctionalized jejunum and his patient had a stormy postoperative course with atelectasis, bronchopneumonia and thrombophlebitis. Following these postoperative complications, the patient recovered without difficulty and was asymptomatic at the time of eleven-month follow-up. A postoperative gastrointestinal series revealed a small amount of barium in the loop of defunctionalized jejunum, but no evidence of reflux reaching the biliary tree. A small amount of air in the biliary tract from the jejunal loop was noted on x-ray and revealed the common duct with the left and right hepatic ducts. A slight cystic dilatation was visible. Archambault et al. [*1*] reported the treatment of a case of choledochal cyst with a Roux-Y type of anastomosis, with a defunctionalized antiperistolic limb of jejunum. The patient had a benign postoperative course, and at the four and a half-month follow-up was asymptomatic. A gastrointestinal series revealed no extension of barium toward the biliary tract even with abdominal pressure and a deliberate attempt to cause reflex of barium.

One other type of anastomosis should be mentioned and that is cholecystoduodenostomy if the cystic duct is patent. This has been utilized with satisfactory results but would seem to be a poor choice when compared to direct anastomosis of the cyst to the gastrointestinal tract inasmuch as cholecystoduodenostomy might result in kinking of the cystic duct with obstruction to the flow of bile, thus defeating its purpose.

CASE REPORT

This forty-three year old white woman was admitted to the hospital on September 3, 1954, with a history of vague upper abdominal gastrointestinal complaints dating back to 1948. At that time she had had an upper gastrointestinal series (Fig. 1) which had revealed no abnormality, and an oral cholecystogram following which the gallbladder had not visualized. The patient was then essentially asymptomatic without specific therapy until four years prior to admission when she experienced acute abdominal pain in the right upper quadrant accompanied by severe nausea and vomiting and followed by clinical jaundice. The attack was severe enough to require hospitalization during which time an oral cholecystogram again failed to reveal visualization of the gallbladder. Following subsidence of the attack, the patient was discharged and remained essentially asymptomatic until one year later when she had another similar episode, including jaundice which subsided approximately one to two weeks after the attack. Since that time the patient has had acute, but less severe, attacks of pain in the right upper quadrant associated with nausea and vomiting, but there have been

FIG. 1. Upper gastrointestinal series performed in October, 1948, showing a circular filling defect from an intraluminal lesion in the second portion of the duodenum. (U. S. Army photograph.)

FIG. 2. Oral cholecystogram taken August 26, 1954, illustrating the patient's cholelithiasis. (U. S. Army photograph.)

no further episodes of jaundice. She stated she had to limit her food in total intake and in character more and more in the months prior to admission, being unable to eat any greasy or highly seasoned foods due to marked nausea and flatulence. In August, 1954, an oral cholecystogram was taken which revealed good concentration of dye and the presence of stones within the gallbladder. (Fig. 2.) She was admitted for cholecystectomy. The past medical history was non-contributory with the exception of a thyroidectomy performed in 1947 for carcinoma of the thyroid gland.

Physical examination revealed a well developed, well nourished white woman in no acute distress. There was no evidence of jaundice and the physical examination was within normal limits with the exception of a well healed thyroidectomy scar. There were no palpable masses in the abdomen and the liver was not palpable. Routine preoperative laboratory studies, including van den Bergh tests, were within normal limits. On September 7, 1954, cholecystectomy was performed with choledochostomy and exploration of the common bile duct. Multiple stones were found in the gallbladder. The common duct appeared to be diffusely and evenly dilated to 1.2 cm. in diameter and the wall appeared thickened. No stones or evidence of stones were found within the common bile duct, and a catheter passed into the duodenum without difficulty and without encountering palpable obstruction. Graduated Bake's dilators up to size No. 8 were passed into the duodenum through the common duct and it was noted that a slight resistance to passage of the larger dilators was felt when the dilator passed the sphincter. T-tube drainage was instituted and the postoperative course was completely benign. On the seventeenth postoperative day a T-tube cholangiogram (Fig. 3) was taken and the extrahepatic biliary system appeared to be within normal limits. The pressure of the intraluminal contents of the common bile duct was measured and found to be 10 cm. of water. The T-tube was removed without incident.

On November 13, 1955, the patient was readmitted to the hospital stating that she had been completely asymptomatic for approximately six months following cholecystectomy,

but that intermittent nausea and vomiting with bloating, belching and heartburn had again developed, definitely associated with intolerance to the same fatty and greasy foods which precipitated the same symptoms prior to cholecystectomy. During this six-month period she had had minimal pain and denied any clay-colored stools or dark urine. An intravenous cholangiogram was taken, as was a gastrointestinal series, both revealing a filling defect in the second portion of the duodenum. In view of these findings, the gastrointestinal series performed in October, 1948 (Fig. 1) was re-evaluated and a similar filling defect in the second portion of the duodenum was seen in retrospect, but it did not appear quite as large or as apparent as in the present studies. Examination of other previous studies, including the postoperative T-tube cholangiogram (Fig. 3), failed to reveal any defect but there was not enough dye present in the duodenum for good visualization. The intravenous cholangiogram, taken in October, 1955 (Fig. 4) revealed excellent visualization of the entire duct system with the hepatic ducts visualizing on the ten-minute film. The common bile duct measured 12 mm. in width at its widest point. No evidence of obstruction or stone was seen. As the dye entered the descending duodenum, a circumscribed filling defect within the lower portion of the duodenum was seen. The upper gastrointestinal series (Fig. 5) demonstrated a normal esophagus and stomach with normal motility and mobility. No extrinsic or intrinsic defects were displayed in the stomach, pylorus or first portion of the duodenum. In the region of the junction of the middle and distal thirds of the descending duodenal loop was a prominent, rather evenly marginated, ovoid, negative filling defect with some ballooning out of the duodenal lumen. The mucosa in this area was intact. Due to the fact that this lesion could be demonstrated on the 1948 gastrointestinal series, a malignant neoplasm was believed to be an unlikely possibility. The preoperative impression was probable intramural benign neoplasm of the second portion of the duodenum. Enterogenous cyst, intraluminal diverticulum and ectopic pancreas were also mentioned as possibilities by the radiologist.

Laboratory examinations were as follows: white blood cell count, 7,500 per cu. mm. with a normal differential; hemoglobin, 15.2 gm. per cent; sedimentation rate, 13; hematocrit, 40; bleeding time, coagulation time and prothrombin times were normal. Serologic tests were negative. van der Bergh test: direct, 0.05 mg. per cent; indirect, 0.2 mg. per cent. Routine urinalysis was normal.

FIG. 3. Postcholecystectomy T-tube cholangiogram taken September 24, 1954, revealing a normal appearing extrahepatic biliary tree. (U. S. Army photograph.)

On November 14, 1955, exploratory laparotomy was performed. The duodenum was mobilized and the second portion of the duodenum was opened with a longitudinal incision, exposing a cystic mass approximately 5 cm. long and 2 to 3 cm. in diameter covered by intact, normal-appearing duodenal mucosa and arising from a pedicle which was just proximal to the ampulla of Vater on the posterior wall of the duodenum. Examination of the common bile duct above the duodenum in the hepatoduodenal ligament revealed it to be within normal size, and palpation of the pancreas was within normal limits. Palpation of the cyst within the lumen of the duodenum revealed the presence of one firm oval mass within the cyst wall, approximately .75 cm. in

FIG. 4. Intravenous cholangiogram taken October 28, 1955, demonstrating the ovoid, smooth intraluminal filling defect in the second portion of the duodenum. This is the study in which the choledochal cyst was discovered. (U. S. Army photograph.)

FIG. 5. Upper gastrointestinal series performed November 1, 1955, definitely outlining an ovoid filling defect in the second portion of the duodenum. (U. S. Army photograph.)

diameter, which was found to represent a gallstone when the cyst was opened. Palpation of the cyst resulted in the escape of bile from the ampulla of Vater with no evidence of resistance. An operative diagnosis was thus made of intraduodenal choledochal cyst or diverticulum. The mucosa overlying the cyst was incised and stripped without difficulty and without any evidence of previous inflammation from the cyst wall. The base of the cyst was developed down to where it was found to join the common bile duct, approximately .5 cm. proximal to the ampulla of Vater. A No. 10 urethral catheter was inserted through the ampulla of Vater and up into the common bile duct with no evidence of obstruction. The cyst was then deliberately opened and an attempt was made to demonstrate the communication between the cyst and the common bile duct. This communication could not be visualized from within the cyst and a small probe was unsuccessfully utilized to attempt to demonstrate the communication. The dissection was therefore continued until the junction between the cyst and the common duct was identified. This area was excised, leaving a defect on the anterior wall of the ampulla approximately 1 cm. in length. On the superior medial aspect of the cyst itself, a second duct was transected as it entered the cyst wall. When it was cut, clear, watery-appearing fluid was seen to escape from its lumen. A polyethylene catheter was inserted into this duct and found to enter directly in the center of the head of the pancreas. It was thus believed that this represented a pancreatic duct, probably the duct of Wirsung. As the point of communication between the cyst and the common bile duct was intraluminal and submucosal, the sphincter of Oddi, the distal ampulla and the redundant duodenal mucosa was excised. When this dissection was completed, the cut end of the pancreatic duct

and the common bile duct maintained their position in the posterior wall of the duodenum with no evidence of retraction. Using interrupted, small, atraumatic® catgut sutures, the mucosal edges were sutured to the periductal tissue in a circular manner, thus leaving the ostia of the ducts opening into the duodenum without obstruction. The No. 10 urethral catheter in the common duct and the polyethylene tube in the pancreatic duct were left in place and were brought out through the anterior wall of the duodenum in the corner of the incision which was closed transversely in layers. Prior to closing the duodenum, an extra drainage hole was cut in the intraluminal course of the urethral catheter so that bile could pass from the common bile duct into the catheter and then into the lumen of the duodenum during the postoperative period while maintaining the splinting action of the catheter.

Postoperatively the patient was maintained on Levin tube gastric suction for forty-eight hours following which she was started on oral feeding. There was never measurable drainage from the pancreatic tube postoperatively, but the catheter in the common bile duct drained approximately 300 cc. of normal-appearing, clear bile daily until the third postoperative day, at which time it was clamped and remained clamped with no difficulty. The postoperative course was completely benign and afebrile. On November 30, 1955, a cholangiogram was taken through the catheter and showed the position of the catheter to be excellent. (Fig. 6.) The patient was discharged from the hospital with the catheter still in place. She remained asymptomatic and was seen as an outpatient on December 12, 1955, at which time the urethral catheter was removed from the common bile duct. Postoperative van den Bergh tests were performed and remained within normal limits. On January 4, 1956, the patient was asymptomatic and the physical examination was within normal limits. There had been minimal drainage for twenty-four hours following removal of the catheter from the common bile duct. An upper gastrointestinal series, performed on January 4, 1956, revealed the duodenal loop to be within normal limits at the site of the previously seen cyst. There was no evidence of reflux of barium into the common bile duct. A postoperative intravenous cholangiogram was considered but the patient had been extremely ill with nausea and vomiting following the previous intravenous cholangiogram and refused to repeat the procedure.

FIG. 6. Cholangiogram taken November 29, 1955, by inserting contrast media through the urethral catheter inserted into the common bile duct. (U. S. Army photograph.)

Microscopic examination of the cystic structure revealed the external surface of the cyst to be covered by a layer of normal-appearing duodenal intestinal epithelium containing a submucosa with a muscularis in several areas. The cyst was lined with a variable type of epithelium. In some areas the epithelium was of an imperfect intestinal type; in other areas it appeared indefinite in type and contained small structures simulating isolated intestinal glands and was lined by columnar epithelium surrounded by fibrous tissue in which lipoid laden macrophages could be seen. In that area the stroma presented a picture similar to that observed in cholesterosis of the gallbladder; in other areas the epithelium was taller, single-layered and supported by delicate fronds of branching fibrous tissue, giving the pattern of that found in the common bile duct.

COMMENTS

Although the typical choledochal cyst presents in the supraduodenal area and is manifest by a localized, circumferential dilatation of the common bile duct, it is believed that the reported case definitely represents a choledochal cyst. This case is unusual in that the cyst was located within the lumen of the duodenum and actually represented a diverticulum of the ampulla of Vater, attached to the common bile duct by a small pedicle, rather than a circumferential dilatation of the common bile duct. In retrospect, it is believed that the treatment of this case was more complicated than necessary and that simple unroofing of the cyst would have promoted adequate intraduodenal drainage without the necessity of excision of a portion of the ampulla and without severing the termination of the pancreatic duct. Shallow et al. [*14*] in reviewing 175 cases, found only two which had extended far enough retroduodenally so that the pancreatic duct opened directly into the cyst. It is believed that this is the first case to be reported in which the choledochal cyst presented and was located completely within the lumen of the duodenum. Keeley [*11*] reported the fourth case to contain gallstones and this is, therefore, the fifth case to have been reported with stones within the cyst. Three cases were found in the literature which were not reported as choledochal cyst, but which may fall into a similar category to the case reported herein. Gordimer et al. [*8*] reported a cystic mass in the lumen of the second portion of the duodenum which measured 4 by 8 cm. and was found to be attached to the posterior wall of the duodenum. There was a pinhead-sized opening at the middle of its right side which drained bile and when probed was found to be the termination of the common bile duct. The cyst was filled with bile and contained twenty-four gallstones. Neither the pancreatic ducts nor the opening from the common bile duct directly into the cyst could be identified. Compression of the cyst did not increase the flow of bile from the opening of the common duct and the cyst could not be emptied. Pathologic examination of the cyst wall revealed it to be lined on both sides with typical and identical duodenal mucosa. The authors thus concluded that this represented an enterogenous cyst of the duodenum. Brooks et al. [*4*] reported a case in a thirty year old white man who had had repeated attacks of epigastric pain, nausea and vomiting since the age of fifteen. When the duodenum was opened, a large polypoid mass was found which was attached to the posterior wall of the descending duodenum and was covered with duodenal mucosa. At the free apex of this mass was a slit-like opening through which bile escaped. When the cyst was opened, the ostia of the common bile duct and the ostia of the duct of Wirsung were demonstrated in the posterior portion of the cyst wall. Microscopically, this cyst was also lined with typical duodenal mucosa and the authors concluded that this was therefore a congenital anomaly of the duodenal wall. Wheeler [*18*] reports the case of a sixty-five year old man with biliary colic in whom a cherry-sized cyst was found within the lumen of the second portion of the duodenum, apparently arising from the intraluminal portion of the common bile duct which was treated with incision with release of a flood of bile. The conclusion drawn was that this might well have represented a choledochocele, but unfortunately there was no pathologic report and no gross description of the mucosal lining.

CONCLUSIONS

1. Choledochal cyst is a rare condition which is probably congenital in origin.
2. The typical clinical triad is that of abdominal pain, abdominal tumor and jaundice.
3. The described roentgen examinations are the most helpful procedures in establishing or confirming a diagnosis preoperatively.
4. Intravenous cholangiograms should be a valuable diagnostic adjunct.
5. The prognosis depends greatly on the correct preoperative or operative diagnosis.
6. The treatment of choice is primary anastomosis between the cyst and the duodenum.
7. The normal pressure demonstrated within the common bile duct, utilizing the post-cholecystectomy T-tube, and the normal ductal pattern with no evidence of obstruction demonstrated on the intravenous cholangiogram add further weight to the theory that these cysts are congenital in origin and not secondary to obstruction.
8. An unusual case of a rare entity has been presented.

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Cancer of the Pancreas

WITH TWO CASE REPORTS OF FIVE-YEAR SURVIVALS

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CANCER of the pancreas is a challenge to modern surgery. Results have been disappointing, and only a relatively few patients have outlived the arbitrary five-year survival period. This paper will record two five-year survivals and will emphasize certain important points in technic.

A critical survey of the situation is also timely inasmuch as little progress has been made since Whipple showed that the operation was feasible in man. Up to the year 1930 there had been sporadic attempts to remove the pancreas, with little success. The report of Whipple, Parsons and Mullins [7] in 1935 was epochal, and the operation as modified by Whipple is still considered the standard procedure by most authorities.

Obviously, in this type of surgery as in others the surgeon is frustrated by the difficulty in obtaining earlier diagnosis. This problem has been reviewed by Cattell [3,4] and others, and elaboration of the subject is not indicated here. I do wish to say, however, that the old description of the symptoms, "the gradual onset of painless jaundice," is no longer tenable. It has been our experience that there may be no jaundice at all, providing the cancer is located in the body or tail of the pancreas. Also, a boring, severe pain in the upper abdomen and back is suggestive of cancer of the pancreas. When x-ray studies and other diagnostic methods have been unrevealing, one should always consider cancer of the pancreas as a possibility. We would also urge the diagnostician to perform duodenal drainage. If blood is obtained in the specimen, cancer is almost certainly present. Also, the absence of pancreatic digestive juices adds to the certainty of the diagnosis. Extreme loss of weight does not usually occur until late in the disease.

The accepted concept of the principles involved in cancer surgery calls for excision of the original tumor together with a wide area of normal tissue and its lymph drainage, keeping all of the tissues in continuity. These ideals obviously cannot always be accomplished, but we should endeavor to follow these rules as closely as possible. With this in mind I reported in detail an operation designed to enlarge the scope of surgery for pancreatic carcinoma. In that article [6] I reported ten cases in which surgery was performed, all seen within the period of one year. Some of these operations should have been considered as only palliative since there was very little hope of cure. In spite of this, two patients have survived five years without any return of cancer. Although it is not my intention to repeat all the details of the operation, I do believe it is important to emphasize certain points.

In my opinion it is necessary to enlarge the scope of surgery. In those cases in which cancer exists in the head of the pancreas and the ducts are enlarged it has been found that cancer cells are present in the pancreatic juices and in the ducts. When partial pancreatectomy is performed, some cancer cells or even a metastatic cancer growth may occur in the portion of the gland which is allowed to remain. One of my cases (Fig. 1) illustrates this point. In this case the tumor was comparatively small and well circumscribed, yet a nodule was found in the tail of the pancreas. (Fig. 2.) More frequent total pancreatectomy is therefore recommended. In those cases in which the cancer is confined to the ampulla, partial pancreatectomy seems sufficient.

Hemorrhage during surgery can be a very formidable complication. Vitamin K should be used liberally prior to surgery. The use of silk ligatures is emphasized. The portal vein is exceedingly thin and must be handled with

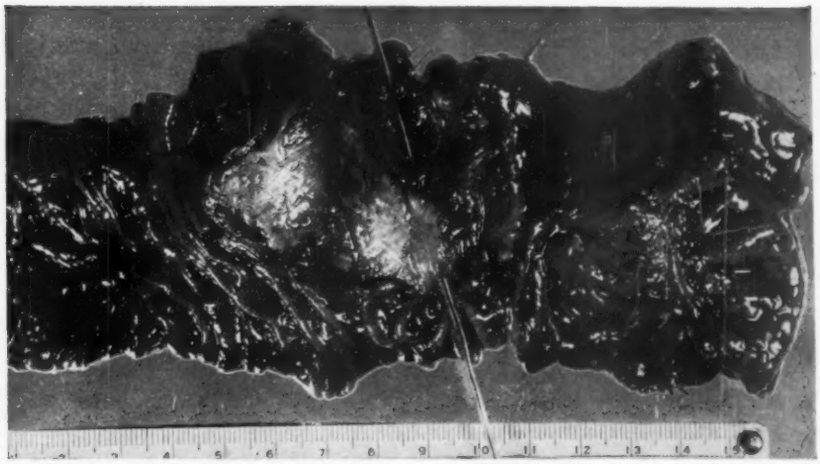

FIG. 1. The duodenum opened. The tumor had bulged into the bowel but had not eroded the mucous membrane. The tumor is shown cut in two. It may be noted that the tumor is relatively small and well circumscribed. The probe is in the common bile duct, which was occluded by external pressure of the tumor. (From: Ross, D. E. *Am. J. Surg.*, 87: 20–33, 1954 [6].)

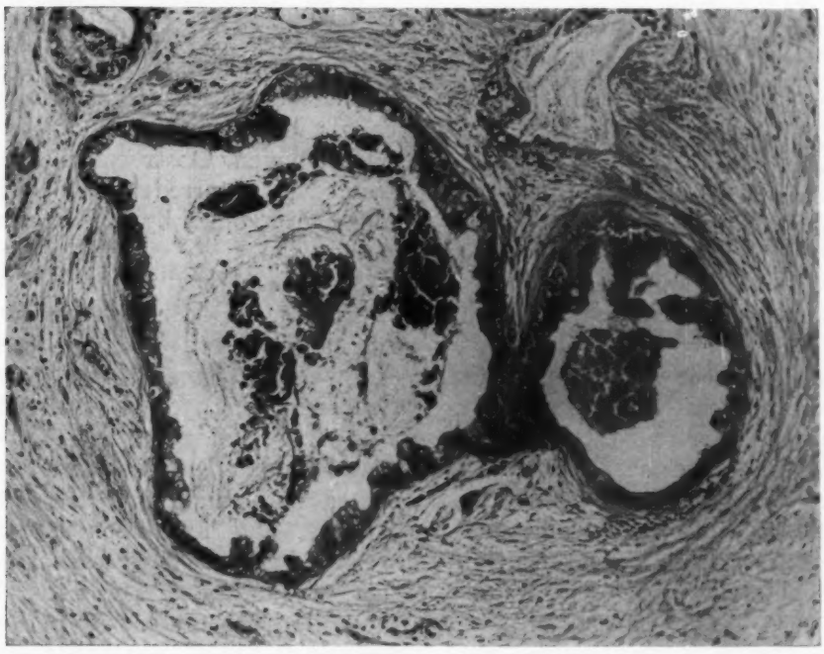

FIG. 2. A nodule was removed from the body of the pancreas at least 5 inches away from the tumor in the head of the pancreas. Section shows a background of rather dense fibrous connective tissue. In the central portion are several irregular groups of epithelial tumor cells which are present within glandular formations. (From: Ross, D. E. *Am. J. Surg.*, 87: 20–33, 1954 [6].)

the greatest delicacy. Blunt dissection cannot be used along the portal vein since this will undoubtedly tear a hole in the vein by pulling on one of the smaller tributaries. Great care should be exercised in tying off the gastroduodenal artery, and sometimes it is advisable to suture the end rather than ligate it.

Where partial pancreatectomy is performed, cutting across this duct may spill the cancer cells into the peritoneal cavity. Brunschwig [2] found residual cancer at the point of section of the common bile ducts in four of twenty-nine cases. It should be obvious therefore that the bile ducts must be removed at a higher

level. For this reason it is my practice to remove the gallbladder together with the common duct and the lymph nodes which are attached, all in continuity with the pancreas. To do this it is necessary to divide the lower end of the common hepatic duct, and this is

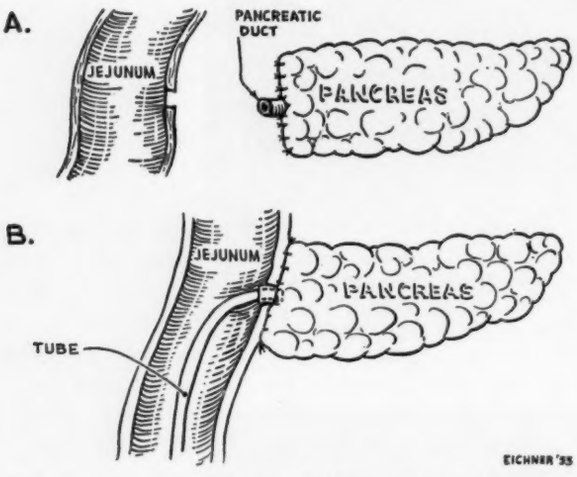

FIG. 3. A, cross section of the jejunum with a small hole cut through the wall. The tail of the pancreas is shown with the pancreatic duct left long and the raw surface of the pancreas closed by silk sutures. B, the duct with the tube pulled through the opening in the jejunum and sutured in place. The capsule of the pancreas is then sutured to the serosa of the bowel.

where anastomosis of the jejunum will be performed.

In order to encompass the maximum amount of possible lymphatic spread it is necessary to remove the spleen and perform total pancreatectomy. In addition, the space between the liver and the upper edge of the pancreas is carefully cleaned to remove the lymph nodes. By performing partial gastrectomy the lymph nodes along both sides of the stomach are also extirpated.

When partial pancreatectomy has been performed, the type of anastomosis of the pancreas to the jejunal loop is illustrated in Figure 3. In cutting through the pancreas the duct is left approximately ¼ inch longer than the end of the pancreas. The pancreatic tissue on either side of the duct is sutured with fine silk. A snugly fitting tube is placed in the pancreatic duct and a small opening is made in the wall of the jejunum, through which the tube and duct are inserted. A silk suture on either side of this hole makes it snug. The capsule of the pancreas is then sutured to the serosa of the bowel. This carries the pancreatic juice a considerable distance down the jejunum, allowing better healing of the anastomosis. Whipple has pointed out how rapidly the pancreatic duct epithelium grows into the wall of the jejunum and blends with the mucosa of the bowel. In some cases I have brought the tube out through a hole in the jejunum by the Witzel enterostomy method and have removed the tube at a later date.

Hunt [5] was the first to anastomose the open end of the pancreas to the jejunum by putting the whole width of the gland into the open end of the jejunum, then sewing the serosa of the bowel to the capsule of the pancreas. I have used this method in many instances and found it satisfactory. However, one of my patients died of a "blow-out" of the end of the jejunum, resulting in jejunal and pancreatic fistula.

It is considered important to bring the jejunal loop up and anterior to the transverse colon. When such a loop is carried through the mesocolon, there is danger of the loop being caught in the raw area from which the pancreas was removed. Obstruction of the bowel from this cause occurred in one of my cases. This danger is avoided by the anterior method.

CASE REPORTS

CASE I. (Queen of Angels Hospital, Los Angeles, No. 10255.) C. G., a fifty-eight year old woman, was admitted to the hospital on September 8, 1950. She had suffered a weight loss of 20 pounds in a five-months period. She had had diarrhea, anorexia and considerable upper abdominal pain for the past three months, and had been jaundiced for two weeks. At surgery an adenocarcinoma of the head of the pancreas was found. Partial pancreatoduodenectomy was performed on September 20, 1950. She made a good recovery and was discharged from the hospital in good condition on October 15th.

On last follow-up, six and a half years after surgery, the patient was free from any recurrence of her malignancy.

CASE II. H. McG. (No. 3727), a fifty-seven year old man, entered the hospital in September, 1950, because of a tumor mass in the left side of his neck. After biopsy, block dissection of the left side of the neck was performed. The pathologic diagnosis was a malignant tumor

metastatic to the lymph nodes. The pathologist could not classify the tumor cells and suggested that they could be of neural origin. Careful examination of the patient failed to reveal the origin of the cancer. However, the patient continued to feel well. He lost 5 pounds in one month and became jaundiced ten days prior

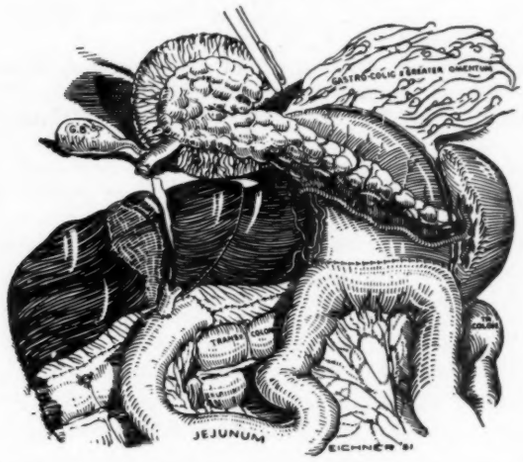

FIG. 4. Shows the various structures removed in the operation of total pancreatoduodenectomy as performed by the author. (From: Ross, D. E. *Am. J. Surg.*, 87: 20–33, 1954 [6].)

to his second entry to the hospital, which was on April 4, 1951. A mass could be felt in the region of the pancreas and the gallbladder was distended. At operation on April 6th we found a tumor which filled most of the head of the pancreas. Lymph nodes were adherent to the head and some of these seemed firm. Other nodes along the pancreas were soft. There were no metastases to the liver or other areas. A complete pancreatoduodenectomy was performed, including the omentum, spleen and two-thirds of the stomach. The pathologic diagnosis was malignant tumor suggesting neural origin of the tumor (neuroblastoma). The nodes adherent to the head of the pancreas showed metastatic carcinoma.

Following surgery the patient was given pancreatic granules and has been maintained on 40 units of insulin per day. His appetite has been moderate and his digestion has been considered good. He has gained approximately 2 pounds each year for six years and seems to be in remarkably good health. A careful check-up at this time shows no evidence of any recurrence.

COMMENTS

It is rather remarkable that in a poorly chosen series of ten cases two patients have survived over five years, with no evidence of recurrence. Admittedly this is a very small number from which to draw conclusions, yet

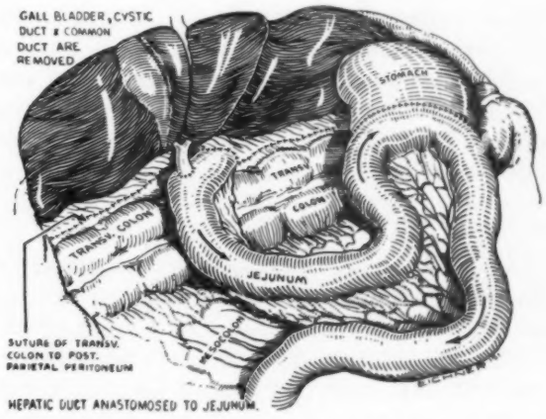
GALL BLADDER, CYSTIC DUCT & COMMON DUCT ARE REMOVED
STOMACH
TRANS. COLON
JEJUNUM
SUTURE OF TRANSV. COLON TO POST. PARIETAL PERITONEUM
HEPATIC DUCT ANASTOMOSED TO JEJUNUM

FIG. 5. Demonstrates the final relationships at the termination of the operation of total pancreatoduodenectomy. (From: Ross, D. E. *Am. J. Surg.*, 87: 20–33, 1954 [6].)

it represents a five-year survival rate of 20 per cent. The obvious lesson to be drawn from this article is that cancer of the pancreas is not as hopeless as some would have us believe. Cattell [3] once intimated that unless better results could be obtained, it might be advisable to abandon the operation. This idea unfortunately permeated the thinking of the profession and has deprived many patients of the possible benefits of surgery. It is my conviction that more hopeful results may be obtained in the future by further research and study, with earlier diagnosis. It seems obvious that the scope of surgery must be enlarged and changed to conform to the recognized standards governing cancer surgery. In a previous article [6] I have given details of my technic of total pancreatectomy. Figure 4 illustrates the structures removed during such an operation. These are the pancreas, duodenum, common bile duct, gallbladder, spleen, two-thirds of the stomach, and the gastrocolic and great omentum. Figure 5 shows the manner in which the anastomoses are completed and the relative position of the viscera. It is extremely important that the stomach be the site of the most distal anastomosis so that food does not regurgitate into the

bile ducts. Also, if some bile enters the stomach, subsequent formation of ulcer will be prevented.

One of the important things to remember is that there is hypersensitivity to insulin in some cases, and that until stabilization occurs the dosage of insulin must be followed with infinite care. In one case of complete pancreatectomy in which the original tumor was quite small the patient suffered two attacks of insulin shock and died undoubtedly as a result of a third insulin reaction. This occurred despite all our pleas for closer observation and control of the patient. Most of our patients require only 40 to 50 units of insulin per day.

SUMMARY

This article emphasizes certain methods of enlarging the scope of surgery. It demonstrates that patients can live and lead a normal life for many years following complete pancreatectomy. At the present time we have five patients upon whom complete pancreatectomy was performed who are still alive and well several years after surgery. There seems to be no physiologic reason why any harmful effect need accrue from total pancreatectomy.

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J. R. Firpo of Argentina describes his experience with two cases of cancer of the breast during pregnancy. He found that the tumors developed rapidly and ulcerated early. There was also a tendency to metastasize rapidly. The author concludes that the treatment of women with cancer of the breast should be the same as for other women in the same age group, irrespective of whether or not the patient is pregnant. Also, when pregnancy is present at an early stage he recommends therapeutic abortion, then surgery, radiotherapy and possibly hormone therapy as the particular surgeon thinks best. When pregnancy is more advanced, he favors prolonging the date of operation and simultaneously protecting the fetus until its chances for survival are better. When pregnancy terminates he recommends institution of whatever other therapeutic measures may be indicated, including removal of both ovaries. In my opinion, bilateral x-ray therapy is equally efficacious. (*Richard A. Leonardo, M.D.*)

Crossed Renal Ectopia

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CROSSED renal ectopia is an uncommon anomaly of the urinary tract, occurring once in every 7,000 autopsies [1]. In order to develop the clinical and radiographic picture of this condition further, a full review of the literature and a report of two cases will be presented.

Crossed renal ectopia is a congenital condition in which a ureter in the normal bladder position crosses the midline to an ectopic kidney lying on the opposite side of the body. This anomaly may be divided into four types (Fig. 1):

Crossed Renal Ectopia with Fusion. This type is the most common of the four. (Fig. 1A.) Abeshouse [2] in 1947 reported four cases and collected forty-seven reported since Wilmer's review [3] in 1938, bringing the total number to 337. Twenty-six cases have been collected since 1947 in addition to thirteen cases published prior to that year [4–34] and not included in the review by Abeshouse. In the absence of definite knowledge of the presence of fusion, our two cases have also been placed in this group. This ectopic form is characterized by fusion of the ectopic and normally positioned kidney. The resulting renal mass varies in anatomic form depending on the portion of the kidneys involved in the fusion. Six different forms of this anomaly have been described. In all these forms axial rotation of both kidneys is impeded with the pelves located anteriorly. (Table 1.)

The most common form is the *unilateral fused kidney with the ectopic kidney inferior.* (Fig. 2A.)

Second in frequency is the *S-shaped or sigmoid kidney.* This varies from the aforementioned in the degree of axial rotation of the ectopic kidney in relationship to the normal kidney. (Fig. 2B.)

The third form is the *lump kidney.* This is relatively uncommon and is characterized by the extensive fusion of the two kidneys resulting in a solid, irregular renal mass. The kidney mass is usually located just above, or at the level of, the sacral promontory. (Fig. 2C.)

TABLE 1
FORMS OF CROSSED ECTOPIA WITH FUSION

Unilateral fused kidney with ectopic kidney inferior	20
S-shaped kidney	11
Lump kidney	6
L-shaped kidney	3
Disc kidney	1
Unilateral fused kidney with ectopic kidney superior	0

Fourth in frequency of occurrence is the *L-shaped kidney* in which fusion occurs between the lower pole of the superior kidney and the upper pole of the transversely positioned ectopic kidney. (Fig. 2D.)

A rare form is the *disc or "doughnut" kidney.* Both kidneys are situated at the same level with their concave surfaces in apposition. Fusion may be only at the superior and inferior poles, or it may involve the entire apposing surface. (Fig. 2E.)

The least common form is the *unilateral fused kidney with the ectopic kidney superior.* Only sixteen such cases were reported up to 1947, and none has been recorded in the literature since. This rare form involves fusion of the lower pole of the superior ectopic kidney with the upper pole of the normally positioned inferior kidney. (Fig. 2F.)

It is interesting to note that of the forty-one cases reported since the review by Abeshouse in 1947, the frequency of occurrence of the six forms closely approximates that of previous reviews. (Table 1.)

Crossed Renal Ectopia without Fusion. This type is much less common than the fused type. (Fig. 1B.) In 1949 Lee [35] collected twenty-

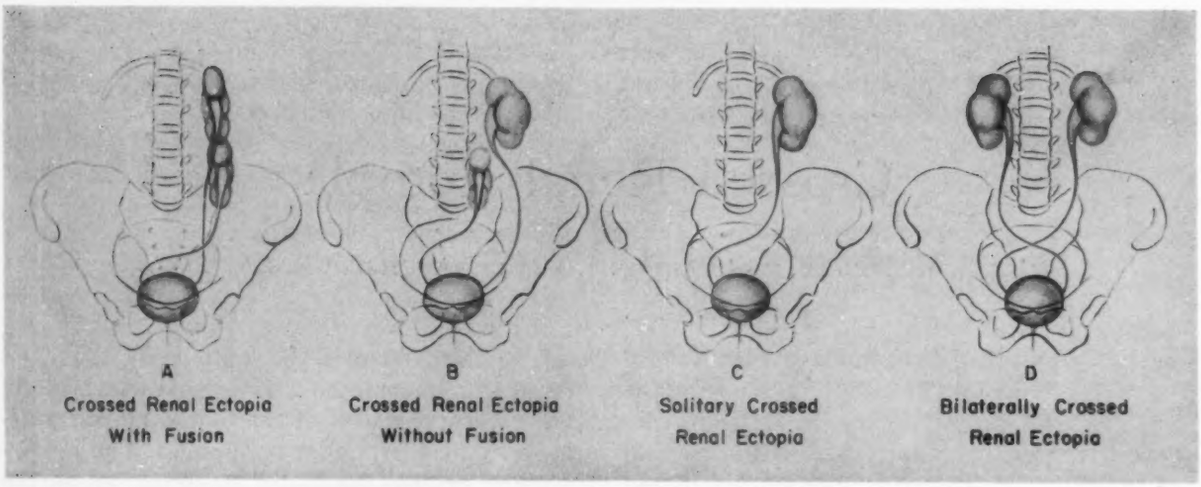

FIG. 1. Four types of crossed renal ectopia.

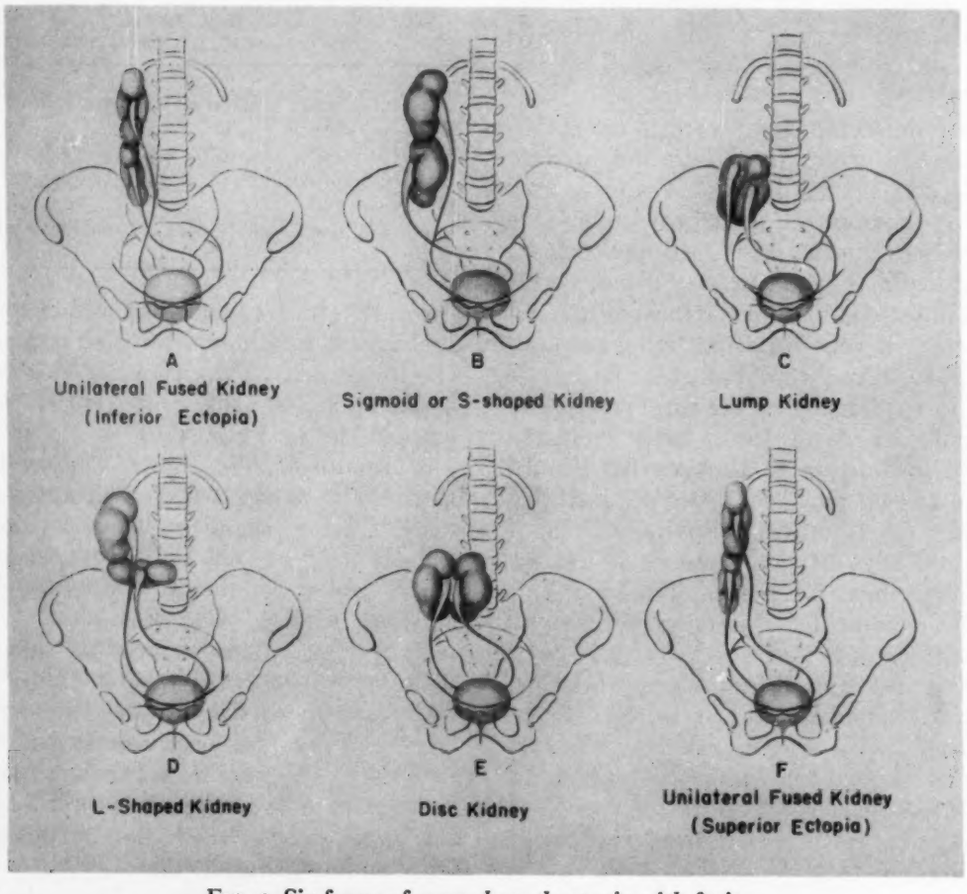

FIG. 2. Six forms of crossed renal ectopia with fusion.

eight cases from the literature and reported one of his own, making a total of twenty-nine cases. Eleven additional cases [9,22,36–44] have been reported since 1949, bringing the total to forty.

Crossed Ectopia of a Solitary Kidney. This type of anomaly is rare. (Fig. 1C.) It is characterized by a congenital absence of one kidney, while the ureter of the remaining kidney crosses the midline to enter the bladder on the opposite side. In 1950 Alexander, King and Fromm [45] recorded the fifth such case.

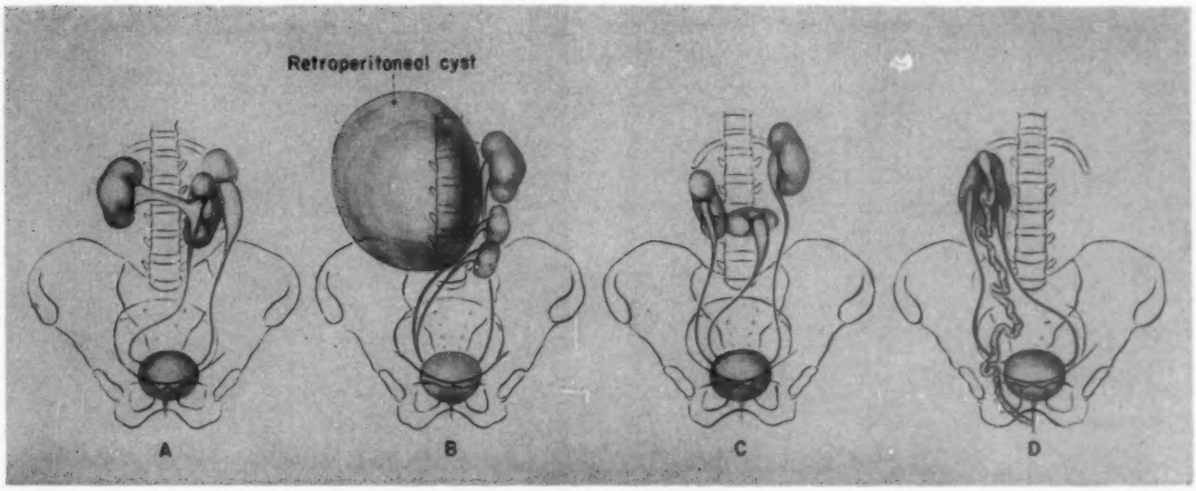

FIG. 3. Artist's conception of three unusual cases of crossed renal ectopia and one case of pseudocrossed renal ectopia (B).

Bilaterally Crossed Renal Ectopia. This ectopic type has been reported only three times. Two cases were collected by Beer and Ferber [*46*] in 1937, and one case was recorded by Wilmer [*3*] in 1938. (Fig. 1D.)

A review of the literature reveals several unusual cases of crossed renal ectopia. In 1949 Potampa, Hyman and Catlow [*17*] reported the case of a fifty-five year old man with a unilateral fused kidney on the left and a crossed ectopic kidney inferior. In the right flank there was a third kidney which was attached to the ectopic kidney by a common pelvis which arched across the midline anterior to the vertebral column. (Fig. 3A.) In 1949 Konrad, Kubik and Pour [*47*] recorded a case of a forty-three year old man with a large right retroperitoneal cyst which, in displacing the right double kidney across the midline to the left side, produced a pseudocrossed renal ectopia. Removal of the cyst allowed the right kidney to return to its normal position. (Fig. 3B.) In 1950 Paoletti [*22*] reported a case of a twenty-four year old man with one kidney in each of the normal positions and a third kidney which lay transversely in a left paravertebral position; the third kidney was fused to the inferior pole of the right kidney with the ureter crossing the midline and emptying into the right side of the bladder. (Fig. 3C.) In 1951 Pia [*26*] reported a case of a nineteen year old girl who had had urinary incontinence all her life. Examination revealed an ectopic ureteral orifice near the urethral meatus and two ureteral orifices in their normal position in the bladder. A retrograde pyelogram demonstrated a lump kidney on the right containing the right kidney, a crossed ectopic left kidney and a supernumerary kidney which was drained by the ureter with the ectopic orifice. (Fig. 3D.)

The embryologic development of crossed renal ectopia has not been clearly established. Many theories have been proposed to explain this abnormal development and are dealt with extensively by Abeshouse and others [*2,3,48*]. Briefly, these include faulty development of the ureteric buds, vascular obstruction to the ascent of the kidneys, and environmental factors involving surrounding tissues and organs.

The incidence of age, sex and location of this anomaly bears mentioning. The ages of patients with this condition vary from a premature fetus to a seventy-nine year old man. The condition was most commonly diagnosed in patients between the ages of twenty-one to thirty, this figure remaining constant whether the diagnosis was made clinically or at autopsy. The sex incidence in all cases reported reveals 220 males and 152 females (sex not given in 51), a male to female ratio of 1.4:1. The renal mass was situated most frequently on the right side, i.e., with the left kidney ectopic. In the cases in which the location was noted, the right side contained the kidneys 222 times and the left side 139 times, a ratio of 1.6:1.

Investigation of the urinary tract in these patients usually reveals some associated pathologic condition. Thompson and Allen [*49*] state

that these patients have only a 3:1 chance of reaching middle-age without developing demonstrable hydronephrosis. In this study forty of fifty-seven patients had some associated pathologic condition. Frequently multiple conditions are present, with the forty patients having sixty-nine pathologic entities in addition to their crossed renal ectopia. (Table II.) Complications involving the crossed ectopic kidney represent 30 per cent of all associated pathologic conditions, whereas congenital deformities of the genitourinary tract comprise 20 per cent of the associated pathologic conditions.

The symptoms of crossed renal ectopia in general depend on the associated pathologic conditions. (Table III.) Pain was the most common complaint, being present in 65 per cent of the cases. An interesting finding was that all patients reporting pain on the side opposite to the renal mass were found to have sigmoid kidneys, and no sigmoid kidneys gave pain on the same side as the renal mass. The only physical finding in this condition was a palpable abdominal mass. Such a mass was present in thirty cases (61 per cent). Pyuria was found in fourteen cases and hematuria in eleven, but these were always in the presence of a complicating urologic disease.

The differential diagnosis includes four other renal anomalies: congenital solitary kidney in which there is compensatory hyperplasia, homolateral double kidney in which the two ureters enter the bladder on the same side, horseshoe kidney in which each kidney pelvis is situated on its normal side, and prevertebral or pelvic renal fusion anomalies with each renal segment situated on its normal side.

TABLE II
ASSOCIATED PATHOLOGIC CONDITIONS (FIFTY-SEVEN PATIENTS)

Ectopic kidney:	
Hydronephrosis	6
Infection	6
Calculus	2
Renal cyst	2
Ureteropelvic obstruction	2
Traumatic necrosis	1
Carcinosarcoma	1
Pyonephrosis	1
Normally positioned kidney:	
Ureteral obstruction	4
Calculus	2
Nephroptosis	2
Hydronephrosis	1
Infection	1
Both kidneys:	
Infarct	1
Hydronephrosis	1
Genitourinary tract:	
Congenital:	
Unilateral renal agenesis	5
Supernumerary kidney	2
Rudimentary ureter	1
Undescended testes	1
Ectopic testicle	1
Inguinal hernia	1
Ectopic ureteral orifice	1
Vesical diverticulum	1
Seminal vesicle agenesis	1
Acquired:	
Cystitis	5
Anterior urethral stricture	1
Urethrorectal fistula	1
Urinary retention	1
Uremia	1
Non–urinary tract pathologic condition:	
Congenital:	
Bilateral dislocated hips	1
Agenesis of sacrum	1
Scoliosis	1
Infection:	
Tuberculosis of spine	2
Pneumonia	1
Malaria	1
Pulmonary tuberculosis	1
Others:	
Hypertension	2
Fracture of skull	1
Colitis	1
Splenomegaly (myeloid leukemia)	1
None	17

TABLE III
SYMPTOMS AND FINDINGS (FORTY-NINE PATIENTS)

Symptoms:	
Pain	32
Dysuria	10
Frequency	9
Chills and fever	5
Weight loss	4
Nausea and vomiting	3
Constipation	3
Nocturia	3
Incontinence	2
Epigastric distress	2
Headache	2
Malaise and fatigue	1
Findings:	
Palpable mass	30
Pyuria	14
Hematuria	11
Tenderness	8
Albuminuria	3
Uremia	1
Anemia	1
Residual urine	1
No symptoms or abnormal physical findings	2

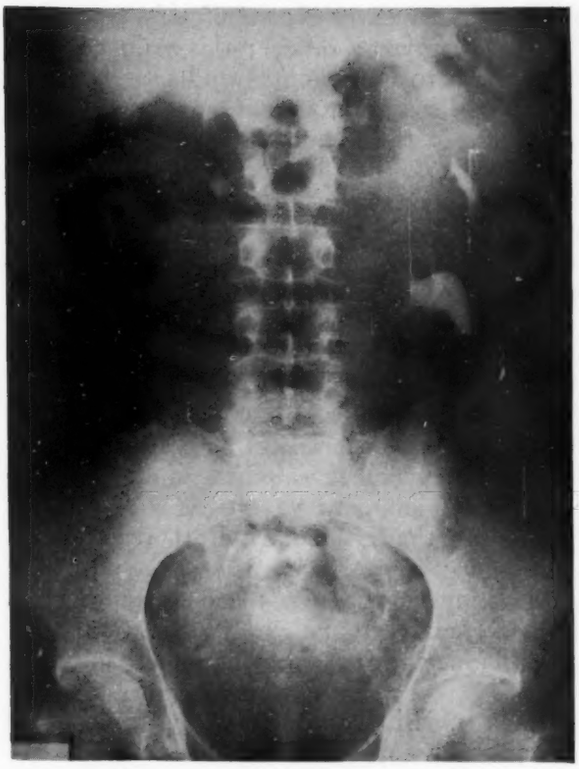

FIG. 4. Case 1. Intravenous urogram showing left crossed renal ectopia.

FIG. 5. Case 1. Retrograde urogram showing crossed renal ectopia. Note path of catheters in the ureters.

There have been a number of reports in the literature in which crossed renal ectopia has not been correctly diagnosed. This condition has been mistaken for nephroptosis, hydronephrosis, renal neoplasm, renal cyst, nephritis, pyelonephritis, pyonephrosis, renal calculi and renal tuberculosis. Crossed renal ectopia has also been diagnosed as various intra-abdominal lesions such as acute or chronic appendicitis, acute cholecystitis and cholelithiasis, intraperitoneal tumors, peritonitis, intestinal obstruction, volvulus, tumor of the liver, tuberculosis of mesenteric lymph nodes and mesenteric adenitis. Incorrect diagnoses of retroperitoneal and pelvic disease have been made, such as retroperitoneal tumor, aortic aneurysm, tuberculosis of the retroperitoneal lymph nodes, uterine fibroma, ovarian cysts and tubo-ovarian disease.

The clinical diagnosis of crossed renal ectopia has become more frequent with the improvement in urologic and radiologic technics. In 1938 Wilmer [3], in an analysis of 286 cases, found that in half the cases diagnosis was made at autopsy. In the present group collected since 1947, in forty-nine of the fifty-seven cases the diagnosis was made clinically. In forty-five cases the diagnosis was made by intravenous pyelography, cystoscopic examination and retrograde pyelography; in the remaining four the diagnosis was made at operation. Retroperitoneal air insufflation was used in two cases and was of advantage in determining

TABLE IV
THERAPY

Medical:	
Infection	9
Hypertension	1
Myeloid leukemia	1
Surgical:	
Nephrectomy	5
Pyelolithotomy	4
Removal of adhesions, ureteropelvic junction	3
Nephropexy of renal mass	2
Resection of renal cysts	1
Resection of supernumerary kidney and ectopic ureter	1
Denervation of renal pelves and pedicles	1
Transposition of ectopic kidney and bilateral nephropexy	1
Ureteroureterostomy	1
No treatment	19

FIG. 6. Case II. Intravenous urogram showing right crossed renal ectopia. Note the path of the left ureter.

the fusion between the ectopic and normal kidneys [31].

The therapy of crossed renal ectopia is usually aimed toward the complications of the condition rather than the treatment of the crossed ectopia itself. In the forty-nine cases with clinical diagnosis in our review, nineteen patients received no therapy, eleven were treated medically and in nineteen various surgical procedures were performed. (Table IV.) Only four surgical procedures were performed because of the crossed ectopia alone. Denervation along both renal pelves and pedicles was performed by Pesqueira and Lopez Engelking [13], with complete alleviation of severe pain. Two nephropexies were performed on the fused renal mass because of pain. One patient continued to have pain but the other became symptom-free. In 1953 Diaz [42] described his operative procedure in a case of crossed ectopia without fusion, in which he transposed the ectopic kidney back to its normal side and performed bilateral nephropexy.

CASE REPORTS

CASE I. L. T., a twenty-five year old white man, was admitted to the Presbyterian Hospital on February 7, 1955, with the complaint of severe pain in his left flank radiating downward to the left scrotum. He gave a history of having had recurring pain of this type since he was ten years of age; this pain recurred every two to four months and usually lasted one day. Physical examination revealed a tender mass in the left upper abdomen approximately 8 cm. in diameter. The prostate and external genitalia were normal. The blood count and urinalysis were normal. Intravenous pyelography revealed an irregular-shaped mass on the left side containing two pelves and no renal shadow on the right. (Fig. 4.) Urine cultures were positive for Escherichia coli. Cystoscopic examination revealed the bladder to be normal and the ureteral orifices to be in their normal positions. Retrograde pyelograms were made which revealed the right ureter to cross the midline to an ectopic kidney lying inferior to the left kidney. Both renal pelves and calyces appeared normal, with the pelves rotated anteriorly. (Fig. 5.) The diagnosis was crossed renal ectopia with fusion (unilateral fused kidney with the ectopic kidney inferior).

CASE II. M. K., a forty-two year old white man, was seen in the office because of frequency, nocturia and dysuria secondary to an upper respiratory infection and furunculosis. The past history revealed a number of attacks similar to the present one. The physical examination was essentially negative for any abnormalities. The prostate and external genitalia were normal. Urinalysis disclosed pyuria. Intravenous pyelography revealed a mass in the region of the right kidney with two pelves and ureters. Both kidney segments showed normal function. There was no kidney shadow on the left. The ureter of the upper kidney on the right extended down into the right side of the bladder. The ureter of the lower pelvis extended across the midline at about the fifth lumbar vertebra and entered the bladder on its left side. (Fig. 6.) The diagnosis was crossed renal ectopia with fusion (unilateral fused kidney with the ectopic kidney inferior) and recurrent cystitis.

SUMMARY AND CONCLUSIONS

1. Crossed renal ectopia with fusion is the most common form of this urinary tract anomaly. Thirty-nine cases have been collected and two reported herein, bringing the total cases in the literature to 378.

2. Crossed renal ectopia without fusion is much less common. Eleven additional cases

collected since 1949 bring the total reported cases to forty.

3. This condition is more common in males, usually involves ectopia of the left kidney and most commonly is diagnosed in patients between the ages of twenty-one and thirty.

4. An associated pathologic condition is the usual reason for clinical investigation in these cases, treatment being directed toward the complicating condition rather than specifically toward the crossed ectopia itself.

5. Pain is the most common symptom and was present in 65 per cent of the cases.

6. All patients reporting pain on the side opposite to the renal mass were found to have the sigmoid form of kidney, and no sigmoid kidney gave pain on the same side as the renal mass.

7. The diagnosis is made by a combination of urologic and radiologic technics including intravenous pyelography, cystoscopic examination, retrograde pyelography and retroperitoneal air insufflation.

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Skeletal Traction in the Management of Certain Problem Fractures

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COMPOUND or comminuted fractures of the long bones and multiple injuries often create complex problems in which the usual closed or open methods of treatment are not advisable. Present day enthusiasm for open reduction and internal fixation in some centers has grown to overshadow and cause neglect of the more simple but effective methods such as skeletal traction. It is our purpose to illustrate the relatively good results which can be gained in treating certain difficult fractures with balanced skeletal traction and early active motion. From the standpoint of complication-free healing and preservation of function the results with this method have proved gratifying and generally better than those that might have been obtained by other means in parallel situations.

The many advantages of this method far outweigh the temporary confinement in bed: (1) Compound wounds may be observed and dressed since the badly injured extremity is free of enveloping encumbrances. (2) Relief of pain and shock are dramatic on application of the traction. (3) Elevation and suspension materially aid the circulation and reduce edema. (4) The reduction may easily be adjusted to correct distraction, angulation and rotation. (5) Optimum length may be preserved in severely comminuted fractures of the femur, although some degree of shortening must be accepted. (6) Early active motion is possible. This tends to settle fragments about joints into position and to preserve function.

Conwell [2] has stated, "I would be very negligent if I did not emphasize the importance of skeletal traction, especially in that type of fracture where other procedures are not indicated or applicable. In many clinics skeletal traction has become a lost art. It is an art that only a good surgeon can appreciate, and it takes as good a surgeon to apply skeletal traction as it does to do operative procedures."

During the past two years at Parks Air Force Base Hospital balanced skeletal traction and early active motion were selected as the treatment of choice or as an adjunct in thirty complex fracture situations which can be classified as follows: (1) severely comminuted and/or compound fractures of the long bones (Cases I, II and III); (2) comminuted fractures involving joint surfaces (Cases IV, V and VI); (3) multiple fractures and fractures associated with injury to the chest or abdomen (Cases I and IV); (4) compound fractures with major nerve, blood vessel or severe skin damage (Case VII).

CASE REPORTS

CASE I. A. H., a thirty-one year old sergeant, sustained bilateral severely comminuted fractures of both femurs (Fig. 1A and B), a compound comminuted T fracture of the tibial plateau (left) and severe shock in an automobile accident on March 5, 1955. Immediate treatment for the fractures consisted of débridement and primary closure of the compound wound. Two days later a plastic spica was employed and the patient was transferred to the definitive care center. Sciatic palsy was present on the right side. Balanced skeletal traction was begun on March 8th with pins through the tibial tubercle (left) and distal femur (right). On March 16th open reduction was performed on the right femur to relieve pressure on the sciatic nerve. Following surgery the traction was continued on both extremities. The compound wound over the tibia healed without incident but the operative wound on the right

1A 1B

FIG. 1A and B. Case 1. Roentgenograph taken shortly after injury. Patient in skeletal traction. A, lateral view of right femur showing large fragment displaced posteriorly; this was responsible for the sciatic palsy. B, anteroposterior view of left femur showing extensive comminution of mid-shaft.

side drained slightly for a time. The sciatic palsy gradually disappeared. By August, 1955, union had progressed to such an extent that the patient could begin ambulation in long-leg braces. When the patient was returned to duty on February 10, 1956 (Fig. 1C and D), eleven months after the original injury, motion at the right knee was 90 to 180 degrees, and at the left 80 to 180 degrees. One and a half inches of shortening of the left femur had been compensated for by a shoe lift and the limp was barely perceptible.

CASE II. R. E., a twenty-two year old airman, sustained a compound (puncture wound) comminuted fracture of the mid-shaft of the left femur due to a bullet wound. Treatment consisted of balanced skeletal traction through the distal femur and early active motion. Ambulation in an ischial weight-bearing brace was permitted after five months. The patient was discharged as fit for duty seven months

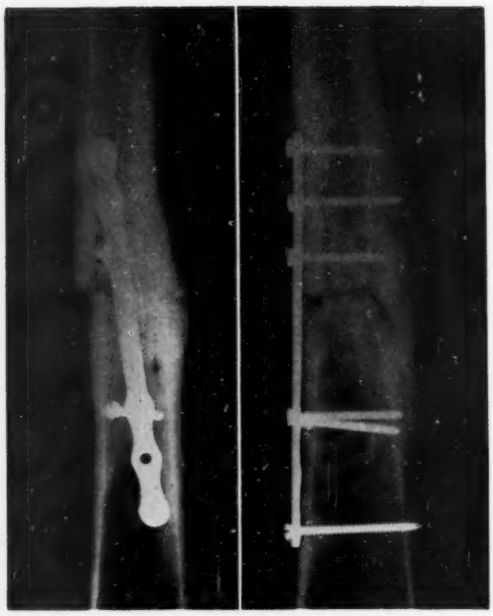

FIG. 1C. Case 1. Anteroposterior and lateral views of right femur eleven months after original injury, prior to patient's return to duty.

after the original injury, at which time he had a full range of motion at the knee joint. There was a slight limp due to the presence of ½ inch of shortening.

CASE III. I. L., a twenty-two year old Philippine seaman, sustained a severely comminuted fracture of the upper one-third of the left femur in an automobile accident on December 22, 1955. After five months of balanced skeletal traction with a pin through the tibial tubercle, bony union had progressed to such an extent that the patient could begin graduated activity. Although the patient was very apprehensive and language difficulty precluded full cooperation, knee motion at the time of release from the hospital, six and a half months after injury, was 90 to 175 degrees and improving steadily. One and a quarter inches of shortening were present. The comminuted fragments were solidly united in good alignment at this time.

CASE IV. A. C., a twenty-five year old airman, sustained a comminuted supracondylar fracture of the left femur (Fig. 2A) associated with multiple rib fractures, bilateral traumatic pneumothorax and severe shock in an automobile accident on July 3, 1955. Treatment of the femur consisted of balanced traction with

FIG. 1D. Case I. Anteroposterior and lateral views of left femur eleven months after original injury. Note over-all good alignment and solid union of fragments.

a pin through the tibial tubercle. Active and passive exercises were begun with the patient in traction one month later, when his general condition permitted. Weight-bearing was begun ten weeks postinjury. Following intensive muscle re-education the patient was returned to duty five months after the original accident; knee motion was from 80 to 180 degrees and there was good quadriceps power. (Fig. 2B.)

CASE V. T. M., a twenty year old airman, sustained a compound (puncture wound) comminuted supracondylar fracture of the right humerus with severe soft tissue swelling and extensive abrasions of the surrounding skin. (Fig. 3A.) Treatment consisted of balanced traction with a pin through the olecranon for five weeks. Nine weeks postinjury the patient was returned to duty; elbow motion was from 70 to 165 degrees, and there was full pronation and supination. (Fig. 3B and C.)

CASE VI. J. C., a thirty-three year old sergeant, sustained a severely comminuted fracture of the lower end of the right tibia and fibula with severe disorganization of the articular surface of the ankle joint in a fall on August 13, 1955. Treatment consisted of balanced skeletal traction with a pin through the

FIG. 2A. Case IV. Anteroposterior and lateral views of right knee showing severely comminuted T fracture prior to reduction.

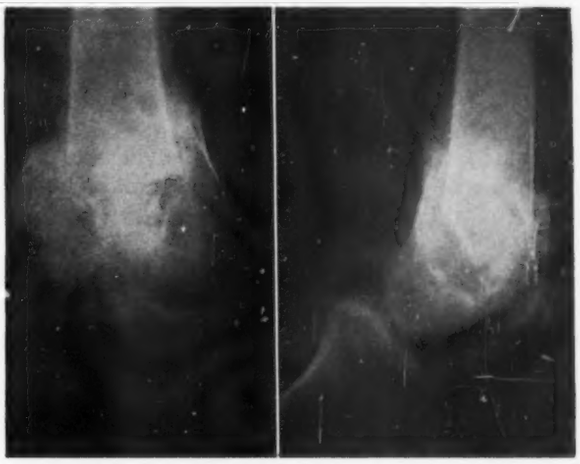

FIG. 2B. Case IV. Anteroposterior and lateral views of right knee five months after original injury, just prior to patient's return to duty, showing union of the fragments in satisfactory alignment.

os calcis and early active motion. On August 27th closed manipulation was performed under anesthesia while the patient was in traction. A walking plaster cast was used for a three-week period after a total of six weeks of traction. The patient was returned to duty three months after the original injury with no pain and ankle motion from 80 to 100 degrees.

CASE VII. R. A., a nineteen year old airman, sustained a compound comminuted fracture of both bones of the right forearm in an automobile accident on October 8, 1954. On his arrival at the hospital the following day there

3A 3B 3C

FIG. 3A to C. Case v. A, oblique view of right elbow shortly after injury. Note extensive comminution (compound) of lower end of humerus. B and C, anteroposterior and lateral views of elbow nine weeks after injury, just prior to patient's return to duty. Note satisfactory position of articular fragments.

was extensive soft tissue damage involving the flexor muscle mass, an absent radial pulse, median nerve hypesthesia and a large laceration of the volar skin. Primary débridement and skin closure were performed. Definitive surgery was postponed because of the extensive soft tissue damage. Balanced skeletal traction was begun with one pin through the distal radius and another through the olecranon for counterpull. On December 15th, with the soft tissues in excellent condition, open reduction and internal fixation were carried out and supplemented by autogenous iliac chips. The operative wound healed *per primam* and solid bony union was present four months later. The patient was returned to duty as a medical corpsman six months after the original injury. At that time he lacked full active flexion of the distal phalanx of the thumb, ring and little fingers, but over-all hand function was good.

COMMENT

Technic of Application. Sterile preparation is done, consisting of a shave and ten-minute pHisoHex scrub. General anesthesia is used only when manipulative reduction or débridement is necessary. Local anesthesia suffices in most other instances and is desirable when severe associated injuries are present. The supracondylar femur, tibial tubercle, olecranon, os calcis and the lower radius are the usual sites for pin insertion. The skin is pulled up and punctured with a No. 11 blade. A Kirschner wire or Steinmann pin usually $\frac{3}{64}$ inch in diameter is then inserted at a right angle to the long axis of the bone. The skin is also pulled up and punctured at the exit site of the pin. Sterile foam rubber pads are threaded over the pin and placed next to the skin, and a spreader is affixed. The limb is then placed in balanced suspension on the appropriate splints and traction is begun. Generally, in an adult 2 to 4 pounds are required for the upper extremity and 7 to 10 pounds for the lower extremity. When reduction is a problem, the weight can be increased initially and reduced at the end of twenty-four hours when a portable roentgenogram has shown satisfactory position and alignment. Further adjustments can be made easily through pads or changes in the line of the splint or traction. Special care must be taken to avoid distraction due to excess weight, as failure to do so may cause non-union or delayed union. Subsequent close supervision is mandatory. Active and passive exercises are started when the patient's general condition permits.

COMPLICATIONS

In the series of cases reported herein no complication of consequence arose which could be attributed directly to skeletal traction. Pin tract infection and injury to a nerve or vessel were not encountered. Small amounts of serous fluid exuded from pin tract wounds in some cases for one or two days following removal.

Superficial skin infection due to maceration occurred in a few patients, but invariably this cleared promptly on removal of the pin and did not involve bone.

One patient complained of mild residual pain at the pin scar and one instance of temporary ligament laxity occurred. Two severely shattered femurs later required bone grafting to fill in or substitute for lost bone substance, but neither could be blamed on distraction due to overpull. Some degree of shortening must be accepted in most badly comminuted diaphyseal fractures. This is necessary in order to obtain enough contact between the fragments to avoid non-union. In this series the maximum shortening was 1½ inches (Case 1).

The lack of recovery of a full range of joint motion in some of the patients was attributable to the severity of injury and subsequent healing reaction. In each instance other methods of treatment involving operative interference or prolonged immobilization in plaster would, in all likelihood, have resulted in further restriction of motion.

SUMMARY AND CONCLUSIONS

A series of complication-fraught, complex fracture problems has been presented to illustrate the relatively good results that may be accomplished in this type of injury through the use of balanced skeletal traction and early active motion. While neither the problems nor the method is new, the latter has been neglected too often recently in favor of more dramatic operative methods with greater risk. In the type of injury in which the usual closed or open methods cannot be applied without danger, balanced skeletal traction usually can be relied upon for complication-free healing and maximum preservation of function. A technic of application has been outlined. Extended hospitalization time comprises the major disadvantage of this method.

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Results of Repair of Large Ventral Hernias with Tantalum Mesh

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LARGE fascial defects have always presented a difficult problem to the surgeon. Over the years many methods and materials have been used to aid the surgeon in repairing large ventral hernias. Since 1945 we have used tantalum mesh or gauze in a number of cases in which it was difficult or impossible to bring the fascia together. Between 1945 and 1953, 2,000 ventral hernias were repaired at the Mayo Clinic. On reviewing these cases we found that tantalum mesh was used in thirty-one. It was thought that a study of the results obtained by this method was indicated.

The utilization of metallic mesh is not at all new to the field of hernia repair. At the turn of the century silver wire and other metals were used in the form of netting. These materials, without doubt, enabled the surgeon to close some hernias that otherwise could not have been closed. According to Throckmorton [4] the use of netting fell into disrepute for several reasons, the most apparent being that the metals being used at that time were too rigid. This apparently gave the patients so much discomfort that many of the meshes had to be removed.

Tantalum, in contrast to stainless steel and vitallium,® is not an alloy but an element. It approaches glass in its resistance to acid and alkali and was originally devised as a dressing for the care of burns. It soon became obvious that it was not too well suited for use in this regard. In 1945 the manufacturers sent some of the mesh to a number of surgeons over the country for study with the idea that it could be used to fill in and repair defects in the human body. No unfavorable tissue reactions were noted and it was well received as an adjunct in surgical care. Koontz and Kimberly [3] and later Koontz [2] alone performed experiments on dogs and rats that revealed many of the characteristics of tantalum mesh. The attributes that have withstood the test of time in human beings are as follows: (1) Tantalum mesh, when in direct contact with bowel, forms only slight adhesions. (2) It does not migrate even when not sutured in place. (3) Tantalum mesh is more resistant to infection than steel. (4) Tantalum mesh implanted in puppies did not cause contraction as the puppies grew. (5) The response to tantalum mesh is a normal fibroblastic tissue reaction and a pliable wound is formed, whereas the response to steel mesh is dense scar tissue which surrounds the mesh rather than infiltrates it. (6) The fibrous tissue starts to grow around and through the mesh immediately after implantation. Under proper conditions this process will be completed after two weeks. Dense tissue will be found after two months.

Many authors speak of "work hardening" and "fracture" of the mesh as bad features of tantalum. It is true that tantalum is subject to work hardening, as are all metals, and fractures do occur. However, we do not believe that these conditions necessarily cause recurrences. All our recurrences happened within the first nine months after implantation of tantalum and none thereafter. We believe that the strength of the repair is in the fibroblastic response around and through the tantalum mesh and not in the mesh itself.

In 1948 Throckmorton [4] presented a series of cases in which tantalum had been utilized to repair hernias complicated by tissue deficiencies. He gave an excellent evaluation of the material. Koontz [1] later reported on a series of 173 cases in which tantalum mesh was

used. At the time of his article it had not been found necessary to remove the mesh in any case. In one case necrosis of the skin had occurred resulting from poor circulation, and the mesh was exposed. Granulation tissue grew through the mesh and the defect was soon closed. Similar cases have since been reported.

STUDY

In a review of problems of this type there are no standard methods for describing the size of the hernias in all their directions or, of course, the quality of tissue available to the surgeon for use.

Of the thirty-one patients who underwent hernial repair with tantalum mesh, twenty-five were considered to be overweight by standard weight tables, the mean average of overweight being 25 pounds. The average age of the group was fifty-eight years. Seventeen of the thirty-one patients had had previous repairs and one patient had undergone five previous attempts at repair. When last heard from three years after repair with tantalum mesh evidence of recurrence had not occurred. The mean average hospitalization was eleven days. Nine of the thirty-one patients were seen as emergency cases with acute intestinal obstruction, and two of these nine patients died during the postoperative period as a result of complications from their obstructions.

As has been mentioned previously, the records of 2,000 patients who underwent repair of ventral hernias between 1945 and 1953 were reviewed. The hernias of thirty-one patients in the group were so severe that they could not be repaired by the usual surgical procedures. This necessitated the use of tantalum mesh, which was used either to cover a very weak repair or to fill in the defect. Twenty-nine of the patients survived the surgical procedure.

An attempt was made to obtain follow-up information on these twenty-nine cases, and information was obtained on twenty-seven. Two patients had died of unrelated causes with no evidence of recurrence of the hernia. The tantalum was removed in two cases, one because of recurrence of the hernia and the other because of persistent drainage. The latter difficulty was found to be due to silk sutures. In seven of the twenty-seven cases recurrences developed. As previously mentioned, all recurrences developed within nine months of the repair. Twenty of the twenty-seven patients or 74.0 per cent are well without recurrence from one to nine years after surgical repair. (Table 1.)

TABLE 1
RESULTS OF USE OF TANTALUM GAUZE IN REPAIR OF SEVERE VENTRAL HERNIAS (TWENTY-SEVEN CASES)

Years Free of Recurrence	No. of Patients	Percentage
2	2	7.4
3	9	33.3
4	4	14.8
5	3	11.1
8	1	3.7
9	1	3.7

SUMMARY AND CONCLUSIONS

Tantalum gauze offers a satisfactory and convenient method of repairing large hernial defects. It does not have the inherent cumbersome nature that cutis grafts and fascial transplants have. It offers no discomfort to the patient that may be considered due to a foreign substance. At the Mayo Clinic 2,000 ventral hernias were repaired between 1945 and 1953. Tantalum mesh was used in thirty-one patients. Follow-up information was obtained in twenty-seven cases. The fact that 74.0 per cent of the group of twenty-seven patients had no recurrence of their hernias was gratifying. Furthermore, it is particularly significant that these results were obtained in a group of patients whose defects were the worst possible, being so severe that no other type of repair seemed feasible.

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The Mid-epigastric, Xiphoid-Excising, Sternum-Splitting Incision

A SURGICAL APPROACH TO THE LOWER MID-THORAX AND UPPER ABDOMEN

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A SURGICAL by-product of World War II was the marked increase in acceptance of the wide utility of the thoracic [2,8] and thoracoabdominal [4,11] approaches for surgery of the lower thorax and upper abdomen. Subsequent experience by surgeons [5,7] has crystallized these gains until now every competent senior surgical resident may choose from a number of acceptable methods the optimum incision for approaching structures lying adjacent to the diaphragm. The multiplicity of such incisions alludes to the failure of one incision completely to satisfy the requirements and restrictions imposed by all lesions and by all surgeons. In utilizing transthoracic and thoracoabdominal incisions the inherent lateral position may decrease already limited cardiopulmonary function [1] and thus preclude their use. The vertical, transverse and oblique abdominal incisions, on the other hand, commonly give such limited exposure of the esophageal hiatus area that the lateral transthoracic approaches are employed in spite of markedly increasing the risk for the patient with marginal cardiopulmonary reserve. If the operator persists in his efforts to limit the incision to the abdomen he may, by reason of limited exposure, be forced to finesse an adequate resection for the surgical care of malignant lesions of the upper gastric area. The resolution of these and of other problems relating to the surgical approach to the lower thorax and upper abdomen is in part provided by the xiphoid-excising, sternum-splitting incision. This incision may be selected in preference to the other thoracic, abdominal or combined approaches, or may be used as a suitable substitute when conditions preclude the use of these latter incisions.

In 1896 von Mikulicz, according to Gottstein [6], made the first recorded thoracoabdominal incision. He started the incision at the umbilicus, continued it to the xiphoid, then laterally to the costal margin and upward, resecting the underlying ribs. Lacking positive pressure anesthesia he carefully preserved the pleural continuity by developing an extrapleural plane for his resection of the lower esophagus and upper stomach. In 1899 he modified the thoracic wall approach to follow the intercostal bundle laterally. In 1897 Milton [9], in the first volume of *Lancet*, described the sternum-splitting approach to the mediastinum and included the transection of the xiphoid as a part of this incision. The approach was designed primarily to explore the mediastinal contents extrapleurally and as such did not enter the peritoneum; however, the lower midline thoracic cavity and the heart in particular was noted to be of easy access. In 1918 Duval [3] reported the combined mid-upper abdominal, xiphoid-incising, sternum-splitting incision and utilized it for exposing the heart and great vessels. The ab-

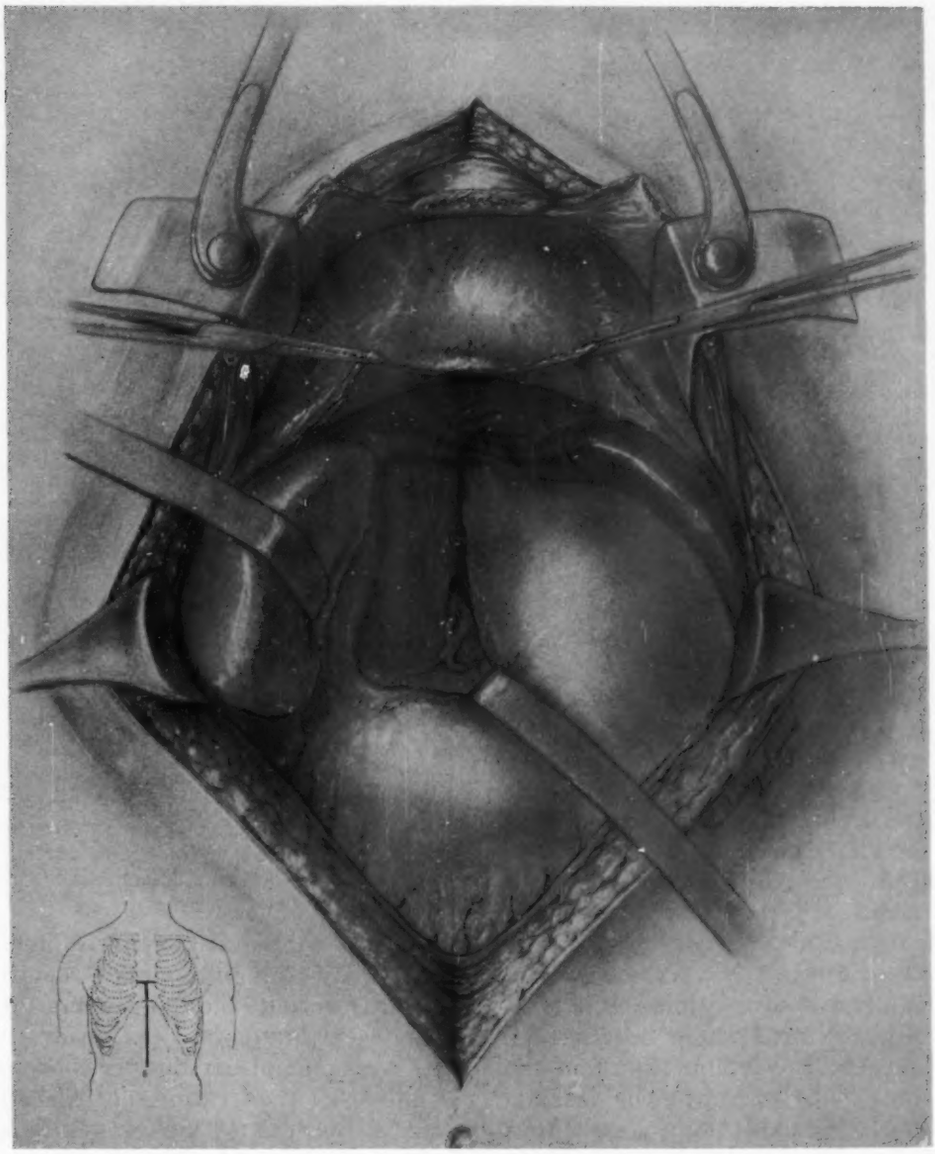

FIG. 1. Xiphoid-excising, sternum-splitting incision showing exposure of lower pericardium and, after mobilization and reflection of left lobe of liver, esophageal hiatus, spleen and stomach. Insert shows extent of incision in sternum and linea alba.

dominal component was added to Milton's incision for better exposure of the inferior and posterior aspects of the heart. Specifically, Duval employed the incision for the removal of a bullet lodged in the pericardium adjacent to the inferior vena cava. In 1951, in a publication of the American Cancer Society devoted to surgery of the upper gastrointestinal tract, Wangensteen [*12*] revived interest in Duval's incision by recommending the addition of left intercostal extensions in resections of the lower esophagus. In 1953 the value of xiphoid excision was re-emphasized by Saint and Braslow [*10*].

TECHNIC

Using endotracheal anesthesia and with the patient in the supine position, implemented by slight elevation of the gallbladder lift, the skin incision is developed (Fig. 1) in the midline from the umbilicus to about the fourth interspace. Initially the linea alba and peritoneum are incised to the xiphoid; abdominal exploration is performed to verify the preoperative diagnosis and in neoplastic disease to determine abdominal resectability and surgical curability. The additional thoracic exposure is effected

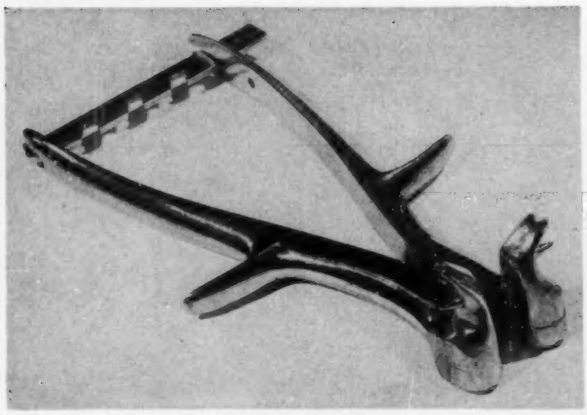

FIG. 2. Sternum retractor of Errington. The instrument lays on the upper sternum. The prongs protrude between the tables of the sternum and fix the retractor as the jaws spread the bone.

only after the lesion has been identified and found operable. The peritoneal incision is extended to the diaphragm which is incised at least to the reflection of the pericardium and occasionally for a distance of several centimeters beyond this attachment. The xiphoid process is excised by scissors or rongeur. Immediately posterior and lateral to the xiphoid, small arterial branches from the internal mammary arteries course from the rectus fascia to supply the subxiphoid area; these arteries are small and well hidden and must be suture-ligated. Using either the Lebsche knife or a rongeur, the sternum is split to about the level of the fourth costal arch and is transected laterally with the same instruments, care being taken at the lateral aspect of the sternum to protect or interrupt the internal mammary vessels. Actually these vessels have only rarely been visualized during this operative approach. Troublesome bleeding from the incised sternum may be controlled by the application of dry gauze under the retractor. It is desirable in incising the sternum to angle the knife from one side to the other in extending cephalad and to describe a curving direction at the same time so that the sternum is cut in a zigzag pattern. This precaution allows for more ready anatomic reconstruction of the sternum without the tendency for over-riding of either edge. The special retractor (Fig. 2) is inserted into the edges of the sternum for wider exposure; manual or self-retaining abdominal retractors are used for the abdominal incision.

During the sternum mobilization moderate care is exercised to prevent bilateral pleural

FIG. 3. Case I. Roentgenogram showing intrathoracic, retrosternal transverse colon and partial pyloric obstruction; barium-filled stomach.

tears and subsequent bilateral pneumothorax. Commonly the left pleural cavity will be entered during the maneuvers around the esophageal hiatus and (as illustrated in Case III) both pleural cavities occasionally must be explored for the treatment of bilateral thoracic lesions. Because the xiphoid-excising, sternum-splitting incision will often be elected as therapy for the patient with marginal cardiopulmonary reserve, the production of even a partial bilateral pneumothorax may be lethal unless recognized and corrected prior to cessation of positive pressure respirations. Residual pneumothorax is aspirated at the time of thoracic reconstruction, and in all cases immediate postoperative semi-recumbent chest roentgenograms must be taken to determine the degree of pulmonary re-expansion. Thoracotomy tube drainage inserted under visual control through the left mid-axillary seventh or eighth interspace is employed only after esophagogastrectomy or after gross contamination of the pleural cavity.

The thoracic component is closed by tran-

sternal sutures of No. 0 twisted wire incorporating space-obliterating bites of the underlying pericardium and diaphragm. The pectoral fascia is repaired with silk. The remaining anterior edges of the diaphragm are sutured to the recti. Figure-of-eight sutures of wire are used to close the linea alba. The skin and subcutaneous layers of the entire wound are repaired with silk.

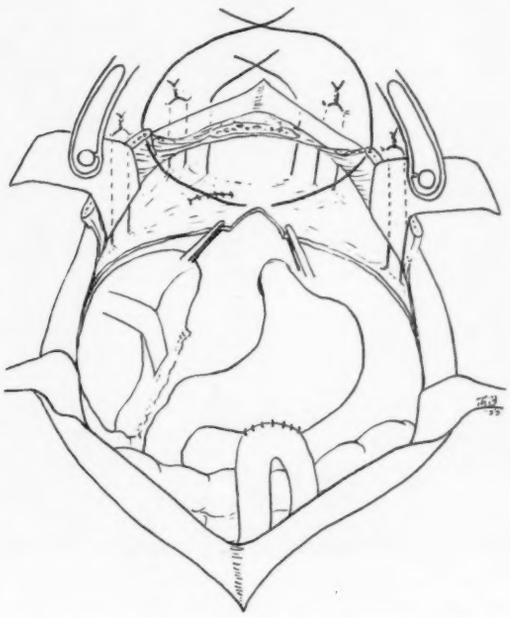

FIG. 4. Schema of retrosternal hernia repair and anterior gastrojejunostomy performed in Case I.

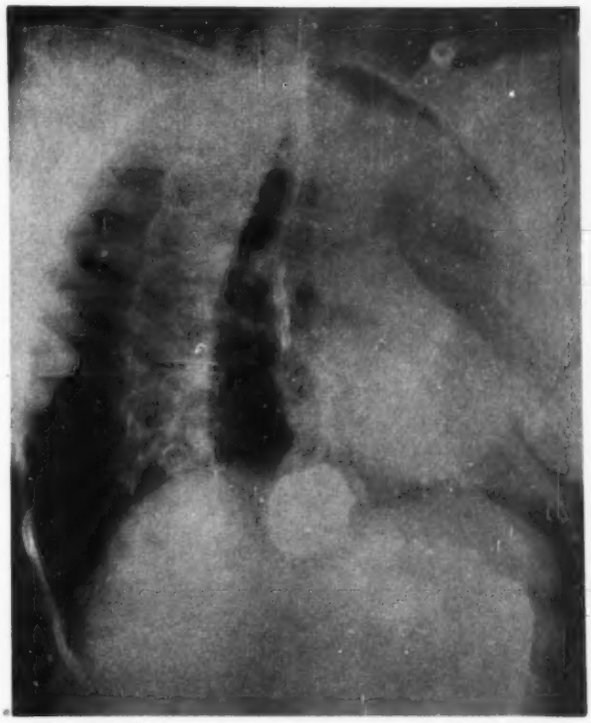

FIG. 5. Case II. Roentgenogram showing esophageal hiatus hernia.

CASE REPORTS

The surgical utility of the incision is reflected in the following case reports.

CASE I. G. K., a seventy-four year old white man, had had pain in the anterior chest and upper abdomen and shortness of breath for five years. He complained of chronic constipation and intermittent vomiting for years; at the time of admission he was unable to retain any food. Gastrointestinal x-ray findings (Fig. 3) revealed that the transverse colon had herniated through the foramen of Morgagni into the anterior thorax and there was almost complete pyloric obstruction.

At operation a mid-epigastric, xiphoid-excising, sternum-splitting incision was made, and mobilization and reduction of the colon from the intrathoracic hernial sac into the abdomen was effected. The sac was repaired (Fig. 4) by wire sutures from the posterior skin through the sternum to the posterior layer of the sac and out to the skin; the sutures were tied. The hernial orifice (9 cm. in diameter) in the retrosternal area was repaired by uniting the anterior edge of the diaphragm to the upper posterior edge of the rectus. Gastrostomy was performed with biopsy identification of the obstructing benign ulcer at the pylorus. Anterior gastrojejunostomy was carried out with wound repair. The patient made an uneventful recovery.

Comment: In an elderly, rather poor risk patient two urgent lesions, one intrathoracic and the other upper abdominal, were readily corrected at one stage through one incision.

CASE II. B. M., a forty-eight year old white woman, had experienced increasing discomfort in the left lower thorax for two years previous to admission. A gastrointestinal x-ray (Fig. 5) revealed an esophageal hiatus hernia. Cholecystogram one year previously had been normal.

At operation a mid-epigastric, xiphoid-excising, sternum-splitting incision was made. Repair of a 4 by 4 cm. hiatus defect was effected.

(Fig. 6.) Cholelithiasis was noted; the smallest calculus was 2 mm. in diameter. The common bile duct was slightly dilated. Operative cholangiography (Fig. 7) revealed a normal extrahepatic biliary system. Cholecystectomy

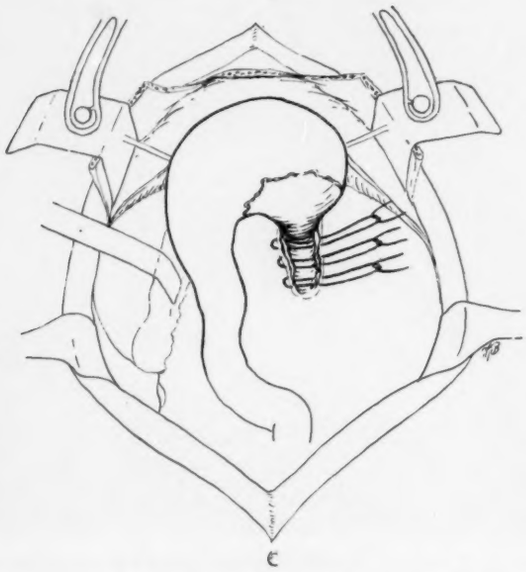

FIG. 6. Schema of diaphragmatic hernioplasty performed in Cases II and III.

FIG. 7. Case II. Operative cholangiogram. Ureteral catheter in cystic duct with injection of 70 per cent urokon® into extrahepatic biliary system and duodenum.

was performed with closure. The patient made an uneventful recovery with satisfactory postoperative repair.

Comment: A symptomatic esophageal hiatus hernia and unexpected cholelithiasis was readily treated at one stage through one incision. Operative cholangiography, routine on this surgical service with all biliary surgery, was made possible through the use of the supine position.

CASE III. F. B., a fifty-nine year old white woman, had experienced tachycardia and pain in the lower anterior thorax for two years prior to admission. Nausea, vomiting and heartburn persisted despite medical management. A gastrointestinal x-ray (Fig. 8) revealed an esophageal hiatus hernia. A chest roentgenogram (Fig. 9) demonstrated a triangular radiopaque mass in the right anteromedial cardiophrenic angle.

At operation a mid-epigastric, xiphoid-excising, sternum-splitting incision was made, and repair (Fig. 6) of the hiatus defect (5 cm. in diameter) was effected. The right pleura was opened retrosternally, with identification of an anterior inferior mediastinal mass of redundant pericardial fat. Closure was performed, and the right thorax was aspirated. A postoperative x-ray revealed evidence of satisfactory repair.

Comment: An esophageal hiatus hernia and an undiagnosed right anterior inferior mediastinal compartment mass were readily treated at one stage through a single abdominothoracic incision.

CASE IV. T. B., a fifty-eight year old Negro man, had experienced increasing dysphagia for four months prior to admission. He had lost 30 pounds in weight. Severe pulmonary emphysema with pulmonary insufficiency was noted. A gastrointestinal x-ray (Fig. 10) gave evidence of obstruction of the lower esophagus with an upper gastric filling defect along the lesser curvature.

Esophagoscopy and biopsy revealed evidence of adenocarcinoma of the lower esophagus. A mid-epigastric, xiphoid-excising, sternum-splitting incision was made and a scirrhous tumor of the mid-lesser curvature of the stomach was found which extended cephalad and involved the lower 5 cm. of the esophagus. Metastatic

FIG. 8. Case III. Roentgenogram showing esophageal hiatus hernia.

FIG. 9. Case III. Chest roentgenogram showing radiopaque mass in right anterior cardiophrenic angle.

FIG. 10. Case IV. Roentgenogram showing narrowing of distal end of esophagus and filling defect of high lesser gastric curvature.

nodes were noted in both curvatures and the inferior pulmonary ligament. Palliative resection of the lower 7 cm. of the esophagus and the upper two-thirds of the stomach was performed with esophagogastrostomy repair. (Fig. 11.) Incidental splenectomy was also done. Closure was effected with left intercostal thoracotomy tube drainage. An uncomplicated convalescence ensued. A postoperative x-ray showed a satisfactory esophagogastric lumen.

Comment: In a pulmonary cripple, palliative esophagogastrectomy with satisfactory reconstruction of the gastrointestinal tract was performed. Because of the accessibility of the inferior pulmonary ligament, the curability of the lesion could be much more accurately assayed at surgery.

COMMENTS

A number of definite advantages as well as limitations accrue through the use of the xiphoid-excising, sternum-splitting incision. In general it is the most simple and bloodless approach to the upper abdomen and lower thorax. Because the patient is placed in the supine position, the incision is best adapted to those persons with restricted cardiopulmonary reserve. The incision is of particular value in patients with suppurative pulmonary disease

who require frequent aspiration by the anesthesiologist and for whom the lateral position is denied because of the danger of drowning the contralateral lung.

At the same time most operative pulmonary complications including atelectasis are minimized by the use of this incision not only because there is no wide opening of the pleural cavity or lung manipulation but also because pain is a minimal postoperative complication. In contrast to rib resection and intercostal thoracoabdominal approaches, parietal pain has been no more common than with midline upper abdominal incisions. The ability of the patient to ventilate deeply and to cough during the early postoperative phase is very little restricted by the sternal component of the incision. Postoperative sternum healing and pain have not been problems.

FIG. 11. Schema of repair after esophagogastrectomy performed in Case IV. Actually the anastomosis retracted above the diaphragm after the reconstruction.

In those patients in whom malignancy either is anticipated or identified by chance at surgery, complete abdominal exploration can be performed through the abdominal portion and the operability and resectability of the lesion determined prior to the addition of the thoracic component. The incision is specifically indicated for the repair of retrosternal hernias. With coexistent multiple lesions involving both the right and left upper abdomen or thorax, the incision should be considered and (as in Cases II and III) may present specific advantages over multiple incisions performed at one or more stages. Surgery for stricture of the esophagocardiac junction, cardiospasm and achalasia may be performed readily through the incision. Upper gastric lesions ideally are approached by the xiphoid-excising, sternum-splitting incision, particularly in those patients with a high-lying stomach or with an acute costal margin overlying the cardia. Esophagoduodenostomy and lesser resections of the stomach with a Billroth I type of reconstruction are easily performed with this approach. The left lobe of the liver may readily be resected or used for an anastomosis to the stomach.

Certain general disadvantages accompany the use of the incision and these include the possibility of not only a unilateral but also, rarely, of a bilateral pneumothorax. In the obese person, who commonly is the recipient of the hiatus hernia, the exposure of the esophageal hiatus may be difficult and require considerable mobilization of the left or right curvature of the stomach as well as continued efforts by two strong-armed assistants. On the other hand, the crura of the diaphragm extending caudad from the esophageal hiatus may be approximated under direct vision after reduction of a hiatus hernia. In addition the esophagus readily may be dislocated anteriorly, if such a procedure is desirable, and the crura repaired posteriorly under excellent vision. In general, the repair of hiatus hernia by the xiphoid-excising, sternum-splitting approach is more effectively although technically more difficultly accomplished than by the transthoracic approach. In surgery for neoplasms of the cardioesophageal junction it must be understood that only a limited extent of the lower esophagus is available. Removal of periesophageal nodes and nodes in the region of the inferior pulmonary ligament and pulmonary hilum is precluded unless an extensive intercostal extension of the incision is carried into the left or right hemithorax. Thus, for lower esophageal surgery, a definite limitation is placed on the extent of resectability for neoplasm.

CONCLUSIONS

1. The xiphoid-excising, sternum-splitting incision provides another effective approach to the structures in the region of the esophagocardiac junction. The incision lends greatest ease of accessibility to the lower retrosternal

area and to the left lobe of the liver and fundus of the stomach. A lesser although adequate exposure is afforded the esophageal hiatus.

2. Ready access is afforded lesions affecting the two lower thoracic quadrants and the corresponding two upper abdominal quadrants, thus permitting multiple lesions of these areas to be treated at one operation.

3. The postoperative course has been marked by few complications. In particular, incisional pain has been minimal in contradistinction to the often severe degree of parietal pain attendant with intercostal and rib-resecting abdominothoracic and transthoracic incisions used in the approach to structures near the esophagocardiac junction.

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Early symptoms of carcinoma of the cecum may be pain, change in bowel habits, blood in the stools or presence of a mass in the right lower abdominal quadrant. The operative mortality rate averages between 12 to 15 per cent. The usual procedure consists of resection of the ascending colon and part of the terminal ileum with establishment of an ileotransverse colostomy. Approximately 25 per cent of these patients die of recurrence with metastases within one year after surgery. The remainder live a variable number of years. Some authorities believe that an exploratory operation is indicated if x-rays are inconclusive in clinically suspicious cases. (*Richard A. Leonardo, M.D.*)

Melanoma Arising in a Café au Lait Spot of Neurofibromatosis

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As pointed out by Allen [*1*], café au lait spots are "essentially equivalent to large freckles both clinically and histologically, with some exceptions in the latter instance." Both lesions are ovoid, flat macules with hyperpigmentation in cells of the basal layers resulting in varying colors, but most often resulting in a light tan color.

The exceptions are most prominently the nevus associations in which there is a varying degree of "junctional change," making that café au lait spot in reality a junctional nevus. Many authorities have now accepted the concept of melanocarcinoma arising almost exclusively from junctional nevi or compound nevi in which there is a junctional element. The following case report may serve only to add

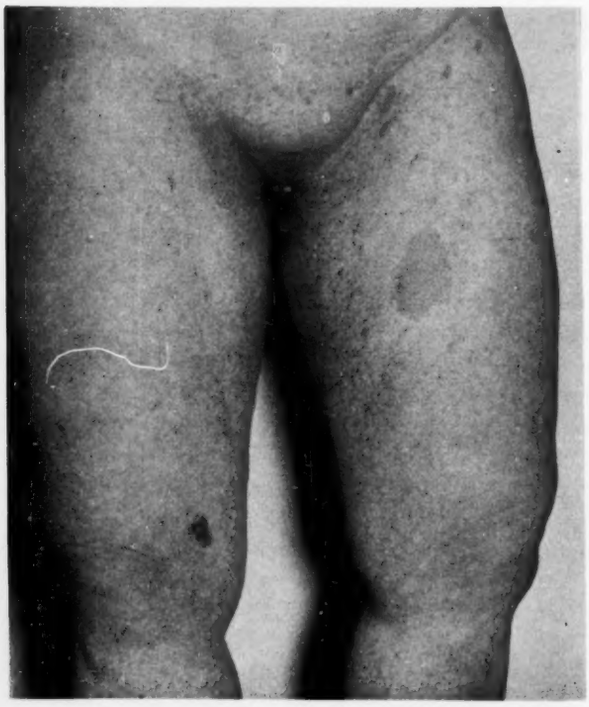

FIG. 1. The neurofibromas are not obvious in this area. The varying-sized café au lait spots are apparent. The malignant lesion on the right thigh is much darker than the others.

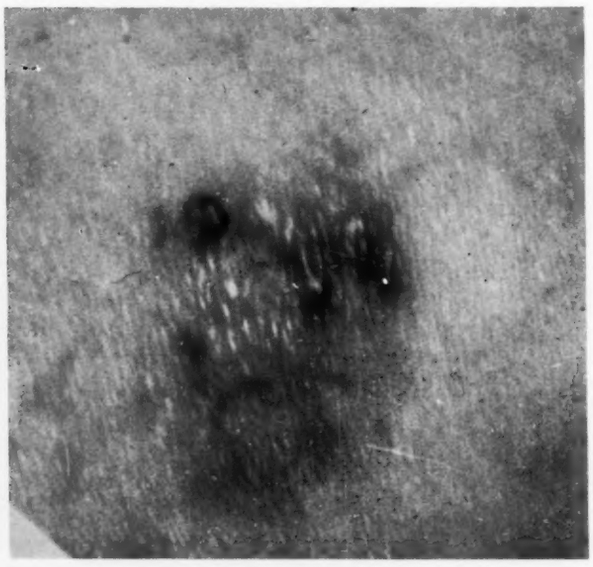

FIG. 2. Enlargement reveals the details of pigmentation and elevation of the melanomatous areas.

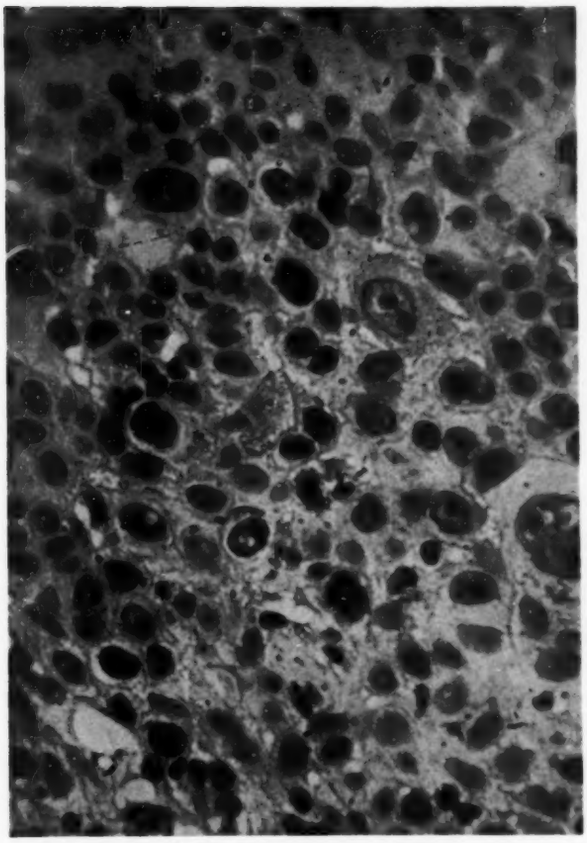

FIG. 3. Section through the original biopsy specimen shows the large anaplastic melanoma cells with included pigment granules. Original magnification ×320.

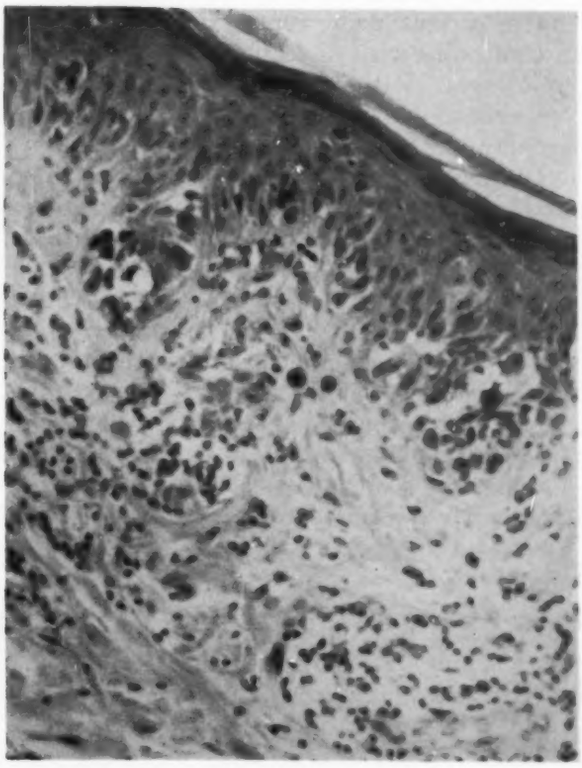

FIG. 4. Section through the café au lait spot of the original tumor demonstrates well the junctional changes. Original magnification ×170.

to the confusion of this issue, but it was believed important to record this case since thorough survey of the medical literature failed to reveal any reports on melanocarcinoma of skin ascribed to a café au lait spot.

CASE REPORT

L. B., a forty-four year old white woman had "freckled" easily all her life and at age thirty had noted multiple small soft elevated nontender nodules of the skin particularly over the face and trunk. One of these had increased to the size of a plum and was excised in another hospital for cosmetic reasons.

These large "freckles" had remained essentially unchanged except that three years prior to admission she had noted one lesion on the right anteromedial thigh to be somewhat darker in color than the many others on the lower extremities. This single lesion allegedly remained the same until nine months ago when she had a rather severe sunburn of the part. After the sunburn healed she noticed the lesion was even more deeply pigmented and had several raised areas within the larger area of pigmentation.

A biopsy specimen taken from the site of the lesion was interpreted as "melanoma," and the patient was referred to Memorial Center in April, 1955. At that time she presented with obvious neurofibromatosis with numerous elevated soft subcutaneous masses from 1 to 10 mm. in diameter. Most of these masses were located on the trunk, neck and upper arms, and were of normal skin color; a few were slightly reddish in color.

There were also innumerable café au lait spots covering the entire skin area with some predominance of the axillae and inner thighs. (Fig. 1.) These spots measured up to 7 by 4 cm. in size and for the most part were light tan in color being contrasted with the normal red-brunette fair complexion of this woman. The exception to this color was noted in a lesion over the medial aspect low on the right thigh in which an area of hyperpigmentation contained several 2 to 3 mm. areas of elevated, more deeply pigmented tissue. (Figs. 1 and 2.)

The patient underwent wide local excision in continuity with dissection of the superficial groin area. Pathologic examination showed "superficial malignant melanoma and negative lymph nodes." (Fig. 3.)

Thorough study of the material revealed further junctional changes (Fig. 4) throughout the excised café au lait spot, and many sections showed an underlying neurofibroma.

Postoperatively the primarily-closed wound sloughed much of its skin edges and grafting was necessary. The patient was readmitted to the hospital in July, 1955, and postage stamp grafting was carried out. While the patient was under anesthesia another café au lait spot which was picked at random from the left groin was excised for study. Microscopic examination of this lesion likewise showed several small areas of junctional change.

SUMMARY

1. A patient with neurofibromatosis and many "café au lait spots" in whom melanocarcinoma developed in one of these spots which contained moderate junctional nevus changes, is presented.

2. It is to be emphasized that the patient in whom this occurs should be reexamined at short intervals to determine malignant change in any of the remaining junctional nevi.

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Traumatic Perforation of the Esophagus Caused by Self-catheterization with Heavy Electric Wire

REPORT OF CASE WITH RECOVERY BY CONSERVATIVE TREATMENT

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TRAUMATIC perforation of the esophagus is a relatively uncommon accident. Self-instrumentation as the cause of perforation is truly unique. An exhaustive search of the world medical literature over a span of the past twenty-five years, including a survey of the rather recent monograph by Thorek [1], has failed to reveal a similar case.

Perforation of the esophagus, regardless of the etiology concerned, is a rather infrequent occurrence. The relatively small number of fifty cases of perforation of the esophagus was observed at the Mayo Clinic by Seybold, Johnson and Leary [2] during the period from January 1907 through June 1949, inclusive. A slightly larger total of sixty-nine patients with esophageal perforation was reported by Jemerin [3] in 1948 from the records of Mount Sinai Hospital, New York City, covering the period from 1925 through 1947. Examination of these two series of cases and contemporaneous medical literature reveal the following pertinent facts: More cases of esophageal perforation are occurring. Recognition of the syndrome has become more widespread throughout the world. Institution of proper treatment with a minimum of delay has effected a marked reduction in mortality.

Traumatic perforation of the esophagus represents an urgent surgical emergency in which the factors of correct diagnosis and time lapse between the accident and surgical intervention are of the essence. The time intervals ensuing between the occurrence of the perforation, correct diagnosis of the condition and surgical treatment are of vital importance to every person so injured. The more quickly surgery is performed, the better the patient's chance for survival. In 1952 Weisel and Raine [4] reported a series of seven cases seen by them over a two-year period. In their series of cases the time interval between injury and diagnosis varied from three to thirty-eight hours. Every patient was treated promptly after diagnosis, by closure of the perforation with drainage of the fascial or pleural space, antibiotic drugs and general supportive therapy. All patients recovered. The report of these authors is most encouraging when we remember that only twenty-five years back, before the advent of the sulfa drugs and later the antibiotics, perforation of the esophagus was considered fatal in practically 100 per cent of cases.

At this point mention should be made of several instances of traumatic perforation of the esophagus in which complete recovery was accomplished by conservative methods. In 1951 Dorsey [5] described the case of a fifty-four year old man who sustained a traumatic perforation of his esophagus following instrumentation. A biopsy specimen was removed from a mass extruding from the left esophageal wall, and perforation unfortunately followed the procedure. Parenteral fluids, penicillin and the application of warm compresses to a large swelling in the left cervical region comprised the main essentials of treatment. The patient's condition rapidly improved under this regimen and he was discharged from the hospital nine days following the removal of the specimen. Seley, in a discussion of Anderson's [6] report pertaining to rupture of the esophagus, discussed the case of a sixty-four year old woman

whose esophagus was ruptured by the passage of an Ewald stomach tube. Intravenous fluids, bilateral pleural taps and a combination of penicillin, aureomycin® and streptomycin administered parenterally produced uneventful recovery in this case.

In 1950 Kernan [7] presented an excellent classification of perforation of the esophagus due to internal trauma. He listed the four principal causes as follows: (1) perforation occurring in connection with the ingestion of foreign bodies and the endoscopic procedures for their removal, (2) perforations occurring during dilation of strictures, (3) perforations occurring during endoscopy to explain dysphagia, hemorrhage or x-ray evidence of esophageal disease, and (4) perforations occurring during the taking of biopsy specimens. In 1943 Paul and Lage [8] described the unusual case of an eighty-one year old white man who died following traumatic perforation of his esophagus caused by the passage of a flexible gastroscope. According to Bisgard and Kerr [9], instrumental perforation of the esophagus is caused in this order of frequency by the following: first the esophagoscope, second the gastroscope and third the stomach tube.

Traumatic perforation of the esophagus undoubtedly occurs with greater frequency than is generally suspected. In this connection it is interesting to note that the syndrome of perforated esophagus is not even mentioned in many current surgical textbooks, nor in numerous publications concerned with the differential diagnosis of surgical emergencies of the chest and abdomen.

In the case described herein the history was most helpful in establishing the correct diagnosis early.

CASE REPORT

S. E., a sixty-three year old white man, was admitted to the Lake View Hospital, Danville, Illinois, at 1 P.M., March 4, 1951. He was obviously in serious condition, as he complained bitterly of severe substernal and epigastric pain, progressive dyspnea and profound weakness.

At 10 A.M. a rather late breakfast had been eaten, which included roast beef. He swallowed a large portion of meat which "stuck in his throat." Attempts to dislodge the meat by coughing and by the cautious ingestion of water were unsuccessful. He then passed a length of heavy electric wire down his throat and into the esophagus in a manner identical to the familiar sword-swallowing act seen at the circus. The bolus of meat was actually pushed down the esophagus and into the stomach by the unusual act of esophageal catheterization. On completion of this unique type of self-instrumentation the patient suddenly experienced severe substernal pain which rapidly extended to the epigastrium. The pain mounted in intensity, becoming excruciating. Dyspnea developed shortly after the onset of the pain and became progressively worse. The patient was soon forced to come to the hospital for relief.

Further interrogation disclosed additional bizarre information. He had the pernicious habit of "bolting his food" and he never took sufficient time to chew his food thoroughly before he swallowed. This habit of faulty eating had existed for as long as he could remember. Up to the time of the present accident he had passed the same length of heavy electric wire down his throat and into the esophagus approximately 200 times over a twenty-year period and had successfully pushed impacted portions of meat into his stomach with no ill effects. The portion of wire used was 17 feet, 8 inches in length and the wire core was slightly blunted at the inserting end. The heavy insulation material which covered the wire core was remarkably well preserved despite the fact that this particular section of wire had been used as an esophageal catheter for a period of twenty years. When not in use it was stored in a loose circular coil. No attempts to sterilize the wire had been made prior to any of its passages into the esophagus.

The family, birth, past medical and surgical histories were irrelevant.

On examination the patient was an elderly, well nourished white man who appeared to be in acute distress. Dyspnea, restlessness, moaning, grunting expirations and cyanosis were all prominent. His temperature was 102.2°F. (oral), respirations 36 per minute and pulse 134 per minute.

The face was pale and the forehead was beaded with perspiration. The lips showed a moderately cyanotic tinge. The pupils were round, regular and equal, and reacted equally to light and accommodation. No icterus, strabismus, nystagmus, exophthalmos or enophthalmos were noted. The nose was normal. The teeth were in poor condition; many were

absent. Dental caries and alveolar pyorrhea were rather extensive. The tongue was heavily coated and the breath was foul. No deviation or tremors of the tongue were noted. The external auditory canals, ear drums and hearing were all normal. The tonsils were small and atrophic. Chronic pharyngitis of a mild degree was seen and a small amount of mucopurulent material was observed in the posterior pharynx. There was no nuchal stiffness and the thyroid gland was not palpable. No cervical adenopathy and no abnormal pulsations were present. A small swelling was observed in the left supraclavicular fossa. This was crepitant upon palpation and due to subcutaneous emphysema. Both lungs showed impairment to percussion posteriorly at the bases and in both lower axillas. The right lower lobe appeared to be involved to a greater extent. Breath sounds were suppressed over the lower portion of the right and left lower lobes, and many rales were heard in these areas. The cardiac sounds were of good quality. No murmurs or arrhythmias were heard. The rate was rapid at 134 beats per minute and the blood pressure was 154/80. The heart showed moderate enlargement to the left, with the left cardiac border located 1 cm. medial to the left anterior axillary line. The skeletal system revealed no abnormalities. Moderate cyanosis of the nail beds was visible. There was no edema of the lower extremities. The patient was well oriented as to time, place and person. His reflexes were physiologic. The cranial nerve system revealed no abnormalities. Rhomberg's sign was not present and there was no slurring of the speech. The skin was warm on palpation, with the exception of the forehead which was cool and covered with droplets of perspiration. No scars, birthmarks or eruptions were noted. No external lymphadenopathy was present. The radial, brachial and temporal arteries were somewhat thickened and slightly tortuous. The genitourinary system and rectum appeared normal.

The history and physical examination of this patient clearly indicated the diagnosis of traumatic perforation of the esophagus, self-induced. Within five minutes after hospitalization a hypodermic injection of ¼ gr. morphine sulfate and $\frac{1}{150}$ gr. atropine sulfate was administered; forty-five minutes later an esophagoscope was passed by the consultant endoscopist. At a distance of 29.5 cm. from the upper teeth a ragged perforation approximately 3 mm. in diameter was observed to involve the right anterolateral esophageal wall. The esophagoscope was then passed to full length and no other pathologic condition was seen. The 7 by 40 bronchoscope was then passed. There were dry crusts in the right main stem bronchus and an exudate was removed by suction from each bronchial tree. The procedure was well tolerated. A chest x-ray was taken prior to endoscopy and revealed moderate cardiac enlargement involving the left ventricle, a small amount of increased density in the lower portion of each lung and a small quantity of gas in the mediastinum at the right border of the trachea. A lateral view of the chest failed to reveal any further abnormality.

The patient endured a rather stormy course for the next eight days. During this period pneumonia developed which involved the right lower lobe and was complicated later by the appearance of empyema of the lower right chest. These conditions subsided, however, and the patient was discharged as much improved on March 29th, twenty-five days after admission.

The area of subcutaneous emphysema at the left supraclavicular fossa had begun to decrease on March 6th and had disappeared entirely by March 9th.

A barium swallow had been performed on March 20th and no evidence of esophageal perforation, ulceration, stricture, diverticulum or stenosis had been observed.

Subsequently the patient was observed on various occasions over a five-year period. He recovered completely from his accident and the subsequent complications of pneumonia and empyema. There have been no further episodes of self-catheterization of the esophagus.

Treatment. Conservative therapy and methods accomplished favorable results in this case. On the day of admission, March 4th, the following was done: At the completion of endoscopy the patient was put to bed in Fowler's position, and oxygen 4 to 5 L./min. was given by face mask as necessary for dyspnea. One-quarter grain morphine sulfate and $\frac{1}{150}$ gr. atropine sulfate, initially administered in the emergency treatment room of the hospital, were given hypodermically when necessary for the relief of severe pain and dyspnea. Penicillin S-R® was started at once with an initial intramuscular dose of 1,000,000 units, and was

then given four hours later in a dosage of 100,000 units by the intramuscular route. This treatment was repeated every four hours until March 15th, when the time between doses was increased to six hours. On March 18th penicillin was discontinued.

Nothing was permitted by mouth on March 4th with the exception of eight 5 gr. bismuth subnitrate tablets which were given in the following manner: one tablet given every thirty minutes, to be well chewed and followed with a small sip of sterile water to facilitate swallowing. One thousand cubic centimeters of 10 per cent glucose in physiologic saline solution was started by vein at 4 P.M. of the day of admission, and this was given slowly. Intravenous feedings, which included amigen® once a day, were continued until March 13th, when the patient was allowed liquids by mouth as desired.

A thoracentesis was performed on March 10th but a dry tap was obtained. Since the patient subsequently improved at a rapid rate both clinically and radiologically, no further chest taps were attempted.

Endoscopy at the time of admission and the thoracentesis six days later were the only surgical procedures performed in the successful management of this case.

Supplemental mutivitamin therapy was maintained continuously. During the intravenous phase of feeding, one ampul Solu-B and 100 mg. vitamin C were added once daily to the morning flask of intravenous fluid. Surbex® was given by mouth, one capsule twice a day, as soon as eating was permitted.

The patient was allowed out of bed on March 15th. The next day he was placed on a soft diet, which he tolerated without any difficulty. Improvement continued so that by March 18th he became ambulatory and was allowed a general diet.

Laboratory Findings. On March 5th the following laboratory work was completed: A complete blood cell count revealed mild leukocytosis of 11,400, with a differential determination of 87 per cent polymorphonuclear leukocytes and 13 per cent lymphocytes. The red blood cells were 4,560,000 per cu. mm. and the hemoglobin content was 12.5 gm. per cent. A specimen of urine was obtained at the completion of an intravenous infusion of 1,000 cc. 5 per cent glucose in physiologic saline. The urine was clear, straw-colored and acid in reaction. The specific gravity was 1.022. No albumin, acetone or blood was present. The reaction for glucose was 4 plus. The microscopic examination showwed no abnormalities. Determinations of non-protein nitrogen and creatinine were normal, 34 and 1.0 mg., respectively.

An electrocardiogram taken on March 10th revealed tachycardia (auricular and ventricular) of 116 beats per minute and also evidence of posterior ischemia. No further abnormalities were noted.

A complete blood cell count was repeated on March 14th and disclosed slight leukocytosis of 12,000 white blood cells per cu. mm. The differential count consisted of 79 per cent polymorphonuclear leukocytes, 18 per cent lymphocytes, 2 per cent eosinophils and 1 per cent mononuclears. The red blood cells were 4,340,000 per cu. mm. and the hemoglobin content was 12.0 gm. per cent. Platelets were adequate.

A third and final complete blood count done on March 24th revealed a normal white blood cell count of 9,200 per cu. mm., with a differential of 75 per cent polymorphonuclear leukocytes, 20 per cent lymphocytes, 3 per cent eosinophils and 2 per cent mononuclears. The red blood cells were 4,520,000 per cu. mm. and the hemoglobin had increased to 12.5 gm. per cent. Platelets were again designated as adequate.

Discharge and Follow-up. As stated previously, the patient was discharged from the hospital in good physical condition on March 29th. Examination of the chest at this time revealed slight impairment to percussion at the right lung base. Slight suppresssion of breath sounds was present at the right lung base posteriorly and a few fine rales were heard in this region and over the lower right axillary area. A period of six weeks' convalescence at home was advised.

The patient was examined on several occasions over a five-year observation period, and had experienced no recurrence of pneumonia, pleurisy, empyema or mediastinitis. When seen on March 15, 1956, approximately five years following his accident, he was enjoying excellent health.

SUMMARY

1. A unique case of traumatic perforation of the esophagus has been described, which

was due to self-catheterization by the passage of a length of heavy electric wire into the esophagus.

2. Prior to the time of the patient's admission to the hospital self-catheterization of the esophagus had been performed at least 200 times over a twenty-year period, with no apparent ill effects.

3. Recovery was successfully accomplished in this case by conservative treatment.

4. Observation of the patient over a five-year period following the perforation revealed no recurrence of the complications of mediastinitis, pleurisy, pleural effusion, empyema or pneumonia. There have been no further repetitions of self-esophageal catheterization.

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Intrathoracic Goiter with Hemorrhage Causing Severe Respiratory Distress

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THE most common cause of respiratory difficulty encountered by patients with goiter is compression of the trachea. The type of compression depends on the size of the goiter and its relation to the trachea [1]. Unilateral enlargement causes a convex deformity of the trachea toward the normal or less affected side. Bilateral and symmetric compression results in a saber-sheath deformity, and compression at different levels brings about an S-shaped deformity. The most common type of goiter causing this pathologic condition is the nodular or adenomatous variety [2], and the usual firm consistency of these nodules adds to the compression. The loss of power of the laryngeal abductor muscles due to the pressure on the inferior laryngeal nerves may be an additional factor [3]. Other workers have pointed out that this is not likely because the slow growth of these tumors causes a gradual stretching of the nerves [4]. Venous engorgement is an important factor when the major portion of the goiter lies in the infraclavicular area. This results in pressure on the innominate veins and superior vena cava. Interestingly enough, most patients with intrathoracic goiter are not conscious of respiratory distress, and only through questioning will the patient be made to realize he has difficulty in breathing, especially in the supine position, on bending or on exertion. The explanation for this is, obviously, the gradual growth of the tumor and the concomitant adjustment to the altered anatomy. Episodes of suffocation are noted by the patient on slight exertion or suddenly at night while sleeping [1–3]. The recumbent position causes congestion of the goiter, which is soon followed with respiratory difficulties. Pillows are of no help in relieving the patient but may further impair breathing because flexion of the head may cause the chin to press on the cervical portion of the enlarged gland and thereby increase tracheal compression. Some of these patients find it necessary to sleep in an armchair. When the patient bends, further respiratory impairment will develop because the goiter is pushed deeper into the mediastinum and, by compressing the veins, increases congestion. Patients with intrathoracic goiter learn by experience to avoid these positions unconsciously.

Sudden death is one of the most tragic and often unexpected endings in cases of intrathoracic goiter. A change in position of the patient's head while he is asleep or an upper respiratory infection that was not considered serious may result in congestion of the substernal goiter, enlarging the gland and completing the tracheal compression to such extent that death may result from suffocation. Sudden hemorrhage within the intrathoracic goiter is another condition which can lead to severe respiratory distress with subsequent death by suffocation [1–5].

Cystic degeneration in patients with adenomatous or nodular goiters is a common and well known occurrence. As mentioned before, the largest percentage of intrathoracic goiters are of this variety. Hemorrhage may be extensive because bleeding occurs in a preformed space without the buffering effect of the firm thyroid tissue.

There are many etiologic reasons for hemorrhage. Besides the usual causes, the effects of changing intrathoracic pressures (i.e., cough, sneeze) enhance the possibility of blood vessel rupture and subsequent bleeding. It is surprising that although the possibility of hemorrhage into an intrathoracic goiter is often mentioned in the literature, only a few such cases are actually reported [5]. The reason for this may very

well be that the patients usually die before medical help can be obtained.

The following case brings out quite dramatically the problems of intrathoracic goiter complicated by massive hemorrhage into its substance.

A. T., a fifty-five year old man in severe respiratory distress, was admitted to the Rochester General Hospital (No. C60677) on December 11, 1955, at 10:30 P.M. The respiratory difficulties had started four days prior to admission, when he was seen by one of us (A. J. P.). At that time he had a temperature of 102°F. and acute septic pharyngitis associated with enlarged, tender anterior cervical lymphadenopathy. Penicillin and other medication were prescribed to relieve his cough which was quite severe at times. Distended veins were noted about the lower portion of the neck; when questioned regarding them the patient stated that he had had a "double goiter" since 1950. Following initial treatment, the pharyngitis improved. During the four days prior to admission, however, he had intermittent bouts of respiratory distress which were aggravated on lying down but were relieved on sitting. On the evening of admission to the hospital his respiratory difficulties suddenly increased to such a degree that immediate hospitalization seemed mandatory. The patient was in severe respiratory distress with marked stridor and sucking in the supraclavicular regions and intercostal spaces with each breath. The respirations were 44 per minute, pulse rate 88 and blood pressure 150/80. He was diaphoretic and no cyanosis was evident. Examination of the neck revealed a diffusely enlarged, non-tender thyroid gland which was rather soft and mushy in consistency. No nodules were palpable on the neck. The anterior and exterior jugular veins were prominent and markedly distended. Auscultation of the lungs revealed only a loud stridor and no rales. The heart sounds were normal, rhythm was regular and no murmur was heard. Because of the prominent dilatation of the veins in the neck, substernal goiter was suspected. An emergency chest film revealed a large intrathoracic mass displacing the trachea to the right and almost completely obstructing it at the level of the third thoracic vertebra. (Fig. 1.) Inferior to the area of obstruction the trachea seemed dilated, while the lungs revealed evidence of acute emphysema. A barium swallow was attempted; however, increasing stridor and rapidly developing suffocation necessitated immediate measures to relieve the tracheal obstruction. By the time the patient was placed on the operating table, he was unable to cough and moist rales could be heard developing over and above the stridor.

FIG. 1. Anteroposterior chest film showing large mediastinal goiter with intrathoracic tracheal obstruction.

Dr. John Dalton, who had been alerted, quickly inserted the endotracheal tube. Fortunately the tube reached beyond the area of obstruction. After the trachea was aspirated, the patient's respirations returned to normal. It was obvious at this time that emergency hemithyroidectomy, including removal of the intrathoracic portion of the thyroid gland, was necessary to relieve this man of the tracheal obstruction. The preoperative diagnosis was nodular goiter with cystic degeneration and hemorrhage into the intrathoracic portion, causing compression and obstruction of the trachea.

Under sodium pentothal®-nitrous oxide-oxygen anesthesia a collar incision was made and the thyroid gland was exposed in the usual manner. The thyroid gland was diffusely

FIG. 2. Gross specimen showing large mediastinal goiter with open cyst at its lower portion.

enlarged and markedly congested, with pronounced distention and dilatation of the veins about the capsule. The blood vessels to the upper and lower poles and lateral portion of the left lobe were ligated and cut. The markedly congested left lobe pushed the trachea with its convexity to the right, rotating it somewhat around its long axis. The isthmic portion could not be discerned with certainty because of the great congestion of the gland obliterating its usual landmarks. Following this the dissection was continued down into the mediastinum. This was carried out almost completely with finger dissection and was aimed at freeing the lowermost portion of the intrathoracic gland. We had to face the problem of being unable to reach the lower pole. Because of this it was decided that mediastinotomy be performed. Although the presence of a hemorrhagic cyst was suspected, the location of this cyst was not determinable by palpation. It seemed unwise to insert a trocar blindly through the thyroid to try to reach a cyst because of the tremendous venous congestion and the possibility of hemorrhage which might have been difficult to control under the circumstances. Thus, while waiting for the bone instruments the effort to free the lower pole of this large mass was continued, and fortunately the index finger punctured a large hemorrhagic cyst. Approximately 150 cc. of large blood clots and liquid blood were evacuated. Following this the removal of the gland was continued without difficulty. The trachea remained in its convex position but to a lesser degree. The neck wound then was closed in the usual manner after leaving a soft rubber drain directed into the residual mediastinal cavity. The endotracheal tube was removed and the patient continued breathing without difficulty.

The specimen (Fig. 2) weighed 270 gm. after evacuation of the blood contained in the cystic area of the mediastinal portion. The mediastinal cyst measured approximately 10 cm. in diameter and was lined with blood clots. Microscopically the gland gave the appearance of a nodular goiter with multiple areas of cystic degeneration.

The postoperative course was uneventful except for transitory cardiac arrhythmia described as paroxysmal auricular flutter. The rhythm reverted to normal twenty-four hours after digitalization. The patient was dismissed on the ninth postoperative day. He was seen by one of us (A. J. P.) six weeks postoperatively. At that time he was comfortable and in no distress whatsoever. Respirations were normal and lungs were clear. The heart had a normal sinus rhythm with tones of a good quality and no murmur. The incision was well healed. The patient was advised to have the right lobe of the thyroid removed some time in the future as an elective procedure.

COMMENTS

An important feature in this case, in addition to the cervical goiter, was the intrathoracic goiter which caused intrathoracic tracheal compression. Under such circumstances tracheotomy would be ineffective as it would be situated above the site of obstruction. A significant diagnostic finding is the engorgement and dilatation of the neck veins. This finding should arouse suspicion as to the presence of a mediastinal mass compressing the large thoracic veins of the upper mediastinum. X-ray examination of the chest with the patient in the anterior, posterior and lateral positions revealed the intrathoracic goiter and the intrathoracic obstruction of the trachea. It was obvious that

emergency thyroidectomy was the only solution to the problem. Time was an important factor and it was fortuitous that the patient was hospitalized in sufficient time to establish diagnosis prior to suffocation.

It is essential that an adequate airway be provided not only as a life-saving measure but also to make appropriate surgery possible. The simplest and most adequate procedure is endotracheal intubation. This solves the problem if the tube passes the area of obstruction. If the tube cannot pass the constricted area, one may try to bronchoscope the patient and insert a catheter through the constricted area, thus establishing an adequate airway [3]. Another emergency measure to be considered is immediate mediastinotomy or at least cutting through the manubrium of the sternum and spreading the bony thoracic inlet in the hope that this will permit fast decompression and the opening of the tracheal obstruction. However, this seems more theoretic than practical, but in an occasional case it may be life-saving.

The operative procedure is best accomplished through a collar incision. Most intrathoracic goiters can be removed in this manner because their blood supply originates in the cervical region. The intrathoracic portion, as in this case, may be quite large, making it difficult to extricate it through a collar incision. Mediastinotomy may have to be performed in order to obtain additional exposure. Ellis et al. [6] believe that this should be performed more often for the removal of intrathoracic goiters. Lahey [4] suggests inserting a trocar and aspirating the intrathoracic goiter through the neck and diminishing its size. However, in cases of acute suffocation the tremendous venous congestion of the gland and its friability would increase the danger of hemorrhage, which may be difficult to control, and so complicate the surgery. Because hemorrhage occurs more often in larger cysts, the blood may be evacuated by puncturing the cyst with the dissecting finger. This procedure may be facilitated by the intraluminal pressure which may cause thinning out of the cyst wall. The collapse of the cyst will then permit the removal of the gland with little or no difficulty.

Intrathoracic goiters should be removed surgically [7]. Removal is indicated not only because of the possibility of malignancy which is more common in this type but also because the goiters may prove fatal by acute suffocation. This outcome can be sudden and unexpected, as the patient may have been asymptomatic until the acute respiratory distress occurred through some complication.

The chances are slight that acute tracheal obstruction will occur in a hospital where highly skilled medical personnel is available. Most of these cases of obstruction are apt to occur while the patient is at home or in some other place where adequate medical help is not available to prevent or correct his sudden complication.

SUMMARY

The causes of tracheal obstruction in intrathoracic goiter have been discussed, with special reference to hemorrhage leading to acute suffocation. A case has been reported of acute hemorrhage into an intrathoracic goiter, leading to marked respiratory distress resulting in near suffocation. Emergency hemithyroidectomy with removal of the intrathoracic goiter led to full recovery of the patient. A plea for the surgical management of intrathoracic goiters has been made.

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Agenesis of the Female Urethra, Trigone and the Anterior Anorectal Wall (A Congenital Anorectourogenital Cloaca)

A NEW PROCEDURE FOR THE CORRECTION OF URINARY INCONTINENCE

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A THOROUGH search of the literature has failed to reveal a single case which was similar in any respect to the case presented herein.

Two cases of urethral absence are cited: Petit's [8] case was that of "a four year old female sans urethra, clitoris and nymphae." The second case, reported by Fraenkel [5], was that of a twenty year old female unable to prevent discharge of urine since childhood. " . . . the vaginal introitus was normal, the hymen was intact and so narrow as not to permit the passage of the tip of the little finger. Immediately above it was an opening with a reddish mucous membrane into which the tip of the little finger could be introduced. From the aperture urine was continuously discharged. At this point there was not, as might be supposed, a deficient urethra, but the bladder opened into the vaginal vestibule." Complete cure was obtained by the technic of Martius using the hymen and the bulbo- and ischiocavernosus muscles.

The following two cases are brought to the reader's attention, although I do not place them in the same category as the aforementioned cases. Langenbeck's [8] case was that of a nineteen year old girl whose " . . . bladder and vagina formed a common canal, gained control afterwards." There is no mention of surgery. This could have been a case of hypospadias. Emmet [4] recorded the case of " . . . a twelve year old female with a cleft of the symphysis pubis and an absence of a portion of the urethra, so that the girl could not retain her urine longer than 15 minutes." There is no mention of the outcome of this case, which was probably an epispadias.

The case to be presented herein is similar to the case of Fraenkel with the additional pathologic condition described by Browne [3] in his 1950 Hunterian Lecture as "a shot-gun perineum."

It is not the intention of this report to discuss the embryology of the alimentary and urogenital tracts, but rather to place on record the only case of a congenital anorectourogenital cloaca and the author's modification of Bricker's [2] application of a loop of the ileum to form an artificial bladder.

CASE REPORT

Because the hospital records of this case were misplaced, the following was acquired from the autopsy protocol.

This patient was a sixty-eight year old Negro woman with a very low I.Q. As a result the clinical and social history is poor and unreliable.

The patient was admitted to the hospital in 1939 for treatment of syphilis and lymphogranuloma venereum. The Frei test and the serologic test for syphilis were positive. She was readmitted in 1943 for treatment of a probable vesicovaginal fistula. Her next admission was in 1949, when complete absence of the urethra was noted. No mention of the anorectogenital defect was made at this or the previous admissions. Surgery was refused on this admission. On the next admission in August, 1953, a diagnosis of congenital absence of the urethra and prolapse of the bladder was made.

On the present admission, November 16, 1953 (I am familiar with only this admission), the patient complained of urinary incontinence of approximately three years' duration; there had been no loss of feces. She had been married for seventeen years and had never been pregnant; intercourse had been normal (?). There had been previous antisyphilitic treatment with arsenicals and bismuth. The past history was otherwise irrelevant except as mentioned on the previous admissions.

On physical examination the patient's blood pressure was 148/72 mm. Hg. Deep reflexes were normal, as were the extremities. There was no adenopathy. Eyes, ears, nose and throat were normal. There were no adventitious sounds in the heart and lungs. The electrocardiogram was within normal limits. Inspection and palpation of the abdomen revealed no abnormalities. Inspection of the external genitalia revealed absence of the perineum with a definite inverted V cleft involving the anus, rectum and vagina for approximately 4 cm. into the vagina. This deformity similated what Denis Browne classified as a shotgun perineum. Urine appeared intermittently. On separating the labia a small, atrophic uterine cervix was seen which was without an anterior vaginal wall; in place of this wall there was a beefy, red tissue which slightly protruded postpubically. From the posterior angles of this tissue at approximately 5 and 7 o'clock there were small apertures (ureteral orifices) from which clear fluid (urine) was intermittently ejaculated. There was no semblance of a urethra or a previous structure present. (Fig. 1.)

The physical findings similated what one would expect to find as the result of complete tears associated with parturition.

As previously stated, the patient had never been pregnant, nor was there a history of injury or undue trauma to the parts involved. Therefore this condition can be classified only as a congenital anorectourogenital cloaca.

The complete blood count was within normal limits; the hemoglobin was 12 gm. per cent and the hematocrit 33 per cent. The serologic test for syphilis was positive. Urea nitrogen was 18 mg. per 100 cc. Admission urinalysis revealed no abnormalities.

On November 17th a posteroanterior roentgenogram of the chest with the patient erect showed several old calcific opacities near the hilum of both lungs. The heart appeared normal in size, shape and position.

On November 20th, the patient was started on a regimen of flo-cillin,® 2 cc. intramuscularly twice daily. Sulfathalidine® therapy, 2 gm. four times a day, was initiated on November

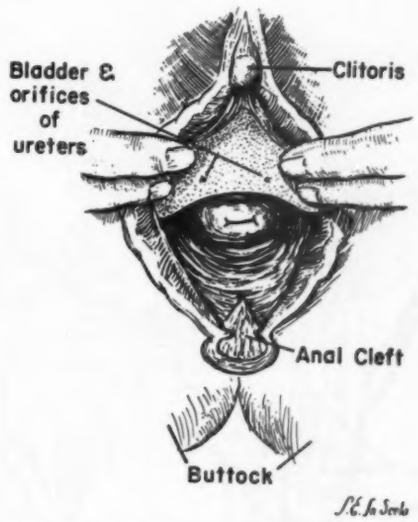

FIG. 1. Congenital anorectourogenital cloaca.

25, 1953. The patient was given a transfusion of 500 cc. of whole blood on November 27, 1953 and on the same day the administration of dihydrostreptomycin, 1 gm. intramuscularly daily, was begun.

The patient underwent surgery on November 30th. After the usual right paramedian abdominal incision, the patient was placed in the Trendelenburg position, and the small bowel and sigmoid colon were placed in the upper part of the abdomen and held there with sterile gauze laparotomy sponges. The ureters were then identified low in the pelvis where they passed through the broad ligaments. The posterior parietal peritoneum with the ureters incorporated was grasped with a pair of Allis forceps near the broad ligaments.

Longitudinal incisions were made in the posterior parietal peritoneum 1 to 1.5 cm. medial and lateral to the ureters near the base of the broad ligaments. The ureter was elevated by passing a pair of curved Kelly forceps posterior to the ureter at the lowermost angles of the incisions in the peritoneum. After the Kelly forceps passed through both points of the incised peritoneum, the strip of peritoneum overlying the ureters was incised transversely at the lowermost angle. The ureter was dis-

sected into the broad ligament, at which point the ureter was grasped with a pair of Kocher forceps and ligated with No. 2 chromic catgut. The ureter was incised transversely just proximal to the point where it had been grasped with the Kocher forceps. The proximal end of the

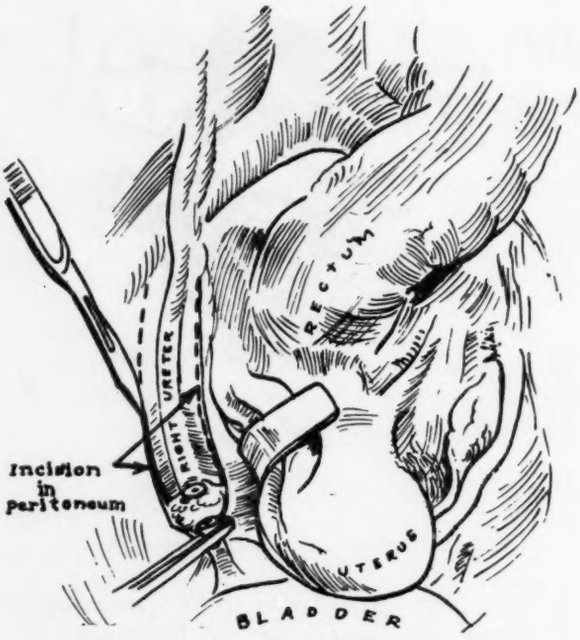

FIG. 2. Exposure of the right ureter.

ureter with the overlying peritoneum was picked up with a pair of Allis forceps in order to eliminate leakage of urine and to avoid trauma to the terminal end of the severed ureter until No. 8 ureteral catheters could be passed following elongation 8 to 10 cm. upward of the incisions in the posterior parietal peritoneum on both sides of the ureters. In this way it was possible to elevate the ureters with the attached peritoneum without disturbing the circulation and innervation. The posterior parietal peritoneum was closed with No. 0000 atraumatic chromic catgut, beginning at the lowermost point of the broad ligament and continuing upward to enclose the ureter completely within the strip of peritoneum; its attachment to the peritoneum was never disturbed. (Figs. 2 and 3.)

After both ureters had been dealt with in this manner, the gauze packs were removed from the abdominal cavity. The terminal ileum was isolated and a section of the bowel measuring approximately 20 cm. in length, as suggested by Rieger and Harris [7], and approximately 20 to 24 cm. from the ileocecal valve, was isolated to construct an artificial bladder employing the author's modification of the Bricker procedure.

After the desired loop of ileum had been acquired between intestinal clamps, the con-

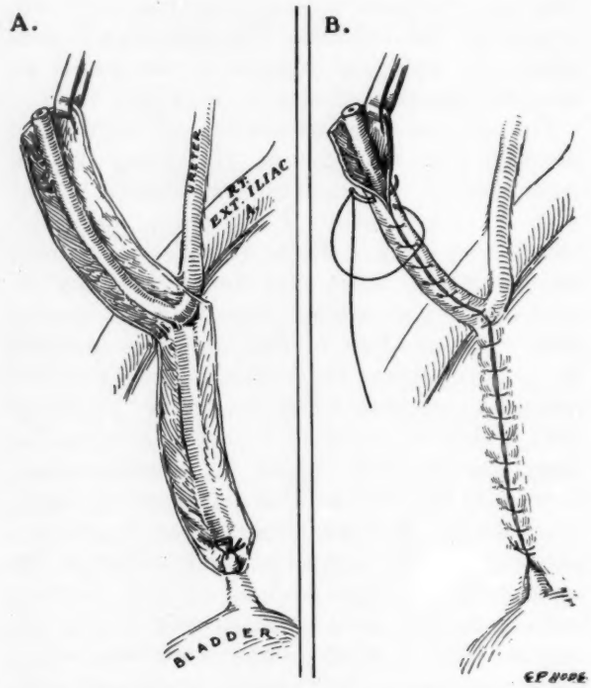

FIG. 3. A, elevation of ureter and attached peritoneum. B, closure of parietal peritoneum with enclosure of the ureter. Allis forceps should grasp the ureter with the peritoneum.

tinuity of the ileum was re-established with a round Murphy button.

A double barrel gun effect was established in the proximal end of the isolated loop of ileum by placing a mattress suture of No. 0 black silk from the convex (free) surface to the mesenteric attachment approximately ½ cm. from the free edge. The urothelium (Maluf [6]) of the right ureter was sutured to the mucosa of the anterior opening and the left ureter to the posterior opening, in the double barrel created in the isolated loop, with interrupted No. 0000 atraumatic chromic catgut. The attached ureters were invaginated into the proximal end of the isolated loop of ileum with interrupted sutures of No. 0 black silk applied to the serosa of the bowel and to the peritoneum enclosing the ureters. The ureteral catheters were left in the ureters and placed through the

isolated loop of the ileum before implantation of the ureters. (Fig. 4.) The distal end of the isolated loop of ileum was brought out to the skin surface through a small incision at McBurney's point. The mucosa was sutured to the skin edge with No. 0000 atraumatic

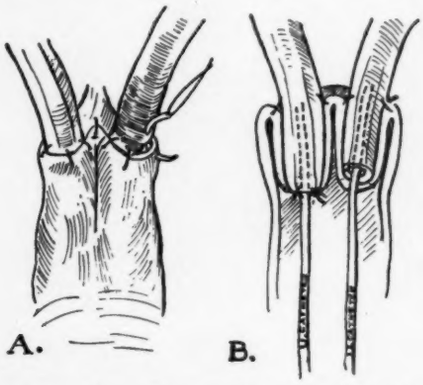

FIG. 4. A, gun barrel closure of proximal ileal loop with attachment of ureters. B, invagination of ureters into ileal loop (false bladder).

chromic catgut. The ureteral catheters were brought to the surface with the isolated loop of ileum and a 22 F. Bardex 5 cc. bag catheter was placed through the external ostium into the artificial bladder to care for any leakage around the ureteral catheters.

The serosa of the ileal loop was attached with interrupted sutures of No. 0 black silk to the peritoneum at its exit from the peritoneal cavity and to the pelvic floor. The opening in the mesentery was closed in like manner and the peritoneal covering of the ureters were sutured to the posterior parietal peritoneum, leaving no apertures for a loop of bowel to enter and eliminating the possibility of an incarceration and strangulation. (Fig. 5.)

The abdomen was closed in layers without drainage and the patient was taken from the operating room in good condition.

During the postoperative course the catheters drained well with satisfactory output. Ureteral catheters were removed after forty-eight hours.

On December 2nd the urea nitrogen was 63 mg. per 100 cc.; carbon dioxide was 35 volumes per cent.

On December 3rd retrograde pyeloureterogram revealed the upper urinary tract to be within normal limits. Heavy metal deposits were noted in both buttocks. (Fig. 6A.) This was the result of previous anti-syphilitic medication with bismuth subsalicylate administered intramuscularly.

On December 4th the patient had some abdominal distention for which a Levin tube was passed through her nose to the stomach. A portable x-ray film of the chest, taken on the

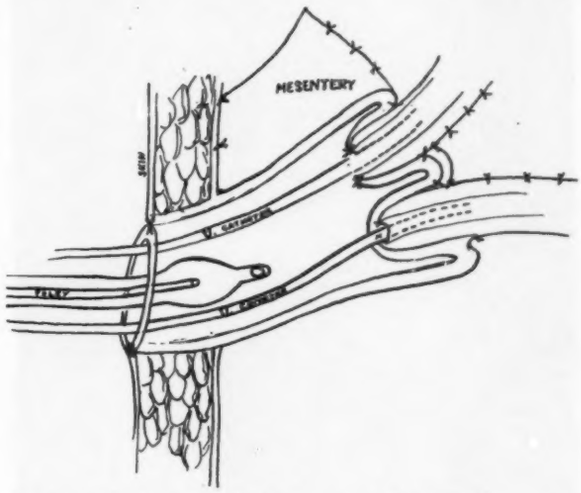

FIG. 5. Distal end of ileal loop attached to skin margin with indwelling catheters.

same day, was not sufficiently satisfactory for interpretation. A portable excretory urogram revealed a dense, spheric opaque shadow 34 mm. in diameter in the right iliac fossa, apparently the anastomosing Murphy button. The Levin tube could be seen in the stomach. The kidneys were not satisfactorily visualized, although there was a good concentration of the opaque material in the artificial bladder ten minutes after intravenous administration. The opaque heavy metal previously mentioned, was present in both buttocks. (Fig. 6B.)

On December 5th there was no improvement in abdominal distention. Blood chemistry showed mild azotemia. Late in the afternoon the patient was returned to surgery for exploration to determine whether or not there was a leak at the point of intestinal anastomosis and/or the ureteral implants. The patient died, supposedly from cardiac arrest, before complete surgical anesthesia was obtained. In spite of morbidity, the abdomen was opened. The peritoneal cavity appeared dry and clean; there was no evidence of leakage, although at the point of anastomosis of the ileum the bowel appeared thinned over the Murphy button.

The autopsy report was as follows: The congenital defect was as stated heretofore; left

6A

FIG. 6A. Retrograde pyeloureterogram taken on December 3, 1953, reveals the upper urinary tract to be within normal limits. Heavy metal deposits can be seen in both buttocks.

6B

FIG. 6B. Portable excretory urogram taken on December 4, 1953, reveals a dense, spheric, opaque shadow 34 mm. in diameter in the right iliac fossa. The kidneys are not satisfactorily visualized although there is good concentration of the opaque material in the artificial bladder ten minutes after intravenous administration. Note opaque heavy metal in both buttocks.

ventricular hypertrophy; pulmonary edema; bilateral pyelonephritis; jaundice; transplanted ureters as described; anastomosis of the proximal and terminal ends of the ileum over a Murphy button (the latter appeared too large for the bowel lumen and the bowel wall appeared partly necrotic).

Microscopic sections confirmed the autopsy findings. Thus the exact cause of death during surgery cannot be determined. The pyelonephritis was suppurative. This no doubt caused severe toxemia, if not septicemia, which would probably account for the jaundice. It is probable that this would have caused death even in the absence of surgery. The role of the surgery in precipitating the pyelonephritis is difficult to assess. It is probable that the patient had had chronic pyelonephritis preceding the surgery. However, one might have anticipated that the antibiotics used to prepare the bowel would have covered the infection. Apparently this was not the case. Perhaps this was due to a resistant organism which was involved. It is probable that if the patient had survived another few days, the bowel would have perforated at the point of anastomosis due to necrosis of the wall because of anoxia caused by the pressure of the too large Murphy button

CONCLUSIONS

I am fully convinced that the ureters can be incorporated in the posterior parietal peritoneum to which they are attached, with preservation of their blood supply and innervation when they are exposed for ureterointestinal implantation. This applies to either the colon or a loop of bowel that is sectioned to be used as an artificial bladder.

The peritoneal covering of the freed ureter supplies adventitia should the operating surgeon desire to invaginate the implanted ureter. There is much less likelihood of a needle entering the lumen of a normal ureter. Aunis and Wells [1] report the implantation of the left ureter in the proximal end of a loop of ileum and the right ureter in the lateral wall.

Azotemia does occur even though 20 cm. (Rieger and Harris) or less of small bowel are used to construct an artificial bladder.

Jaundice in this patient was detected during postmortem examination. The sclerae should be inspected as a routine measure regardless of the patient's race or color.

SUMMARY

A report is presented of an only case of agenesis of the urethra, trigone and anorectovaginal walls (shotgun perineum and an urogenital cloaca (congenital)).

Also presented is a new procedure for exposing the ureters without disturbing their blood supply and innervation by not detaching them from the posterior parietal peritoneum.

A modification of the Bricker technic of surgically making an artificial bladder from a loop of ileum by implanting the ureters in a double barrel effected in the proximal end of the freed loop of ileum has been employed.

Death occurred before an attempt could be made to explore the abdomen five days after the original operation.

Acknowledgment: I wish to express my appreciation to the members of the Medical Art Department of Louisiana State University for their generous cooperation and time spent in the preparation of the drawings and photographs.

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Ruckensteiner's studies in Germany have convinced him that radioactive iodine alone may be valuable in only approximately 10 per cent of cases of thyroid malignancy but that surgery and/or x-ray therapy is also needed in the vast majority of cases. In short, radioactive iodine is a helpful supplementary procedure but is not a substitute for surgery or radiation. In fact the author positively states that radioactive iodine effects only a palliative improvement but not a cure in cases of thyroid malignancy. Hence he recommends its use in inoperable cases or postoperatively to try to reduce the rate of recurrences in thyroidectomy for cancer of this organ. (*Richard A. Leonardo, M.D.*)

Ectopic Pregnancy (Abdominal) with a Living Fetus That Survived

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A twenty-seven year old married Negro woman was admitted to the Nyack Hospital on January 3, 1956. She complained of abdominal pain in both lower quadrants of considerable severity and of one day duration. She stated that she had had similar pains intermittently in the same regions for the past several months but that they were not as marked as the pains she had experienced during the past twenty-four hours. Except for her menstrual history the past history was entirely irrelevent. She had been married five years and her only pregnancy aborted spontaneously after three months' gestation in December, 1953. On the present admission her rectal temperature was 98.6 F., pulse, 110; respiration, 22. A catheterized urine specimen was reported with a specific gravity of 1.013, albumin and sugar were negative. Microscopic examination revealed an occasional white blood cell, occasional red blood cell and an occasional renal epithelial cell. The blood count was: red blood cells 4,250,000 per cu. mm.; hemoglobin, 11.9 gm. per cent; hematocrit, 38 per cent; white blood cells, 11,200 per cu. mm.; polys, 62 per cent; stab cells, 4 per cent; eosinophils, 3 per cent; monocytes, 6 per cent; and lymphocytes, 25 per cent. The venereal screening test was negative for syphilis. The patient stated that her last menstrual period began on June 18, 1955, and lasted four days. On obstetric consultation the abdomen was found enlarged as for a seven months' pregnancy. The fetal heart was audible. On pelvic examination the fornices were clear with no areas of tenderness. The cervix was moderately soft but closed. The impression was that the pain was probably due to an old pelvic inflammatory disease. On surgical consultation it was believed that there was no acute surgical condition within the abdomen. The surgical findings concurred with the obstetric findings. It was believed that the pain was probably due to the expanding uterus drawing on adhesions produced by old pelvic inflammatory disease. A conservative handling of the case was advised and the patient was discharged.

The patient was readmitted to the hospital on January 17, 1956. She gave a history of having been awakened at 4:15 A.M. by sharp abdominal pains in the lower right quadrant and vomiting. She vomited several times during the morning and the pain persisted. Her bowels had moved normally at 6:00 A.M. but not since that time. She was admitted to the hospital in the early afternoon. Her rectal temperature on admission was 99.6 F.; pulse, 108; respiration, 26. A catheterized urine specimen was examined and it showed a specific gravity of 1.019. It was amber in color and slightly acid; albumin and sugar were negative. On microscopic examination a few red blood cells were found. The blood count showed red blood cells, 4,200,000 per cu. mm.; hemoglobin, 11.3 gm. per cent; hematocrit, 36 per cent; white blood cells 16,900 per cu. mm.; segmented polys, 76 per cent; stab cells, 7 per cent; lymphocytes, 10 per cent; monocytes, 7 per cent. Screening test for venereal disease was negative. When I saw her at 6:00 P.M. she lay flat in bed guarding her right side. She did not appear acutely ill but seemed to be in pain. On examination abdominally a mass filled the lower abdomen to a point about 3 fingers above the umbilicus. The fetal heart sounds were audible and the fetal movements were active. There was considerable pain in the abdomen on pressure, rigidity and rebound tenderness most marked at a point about an inch above and just medial to the anterior superior iliac spine on the right side. The remainder of the abdomen was soft and tender. On vaginal examination the cervix

was closed and somewhat soft. The mass filling the lower abdomen was deemed to be the pregnant uterus. Fornices contained no masses and were not tender.

A diagnosis of acute appendicitis was made and it was decided to perform an immediate laparotomy.

Under spinal anesthesia a low left oblique incision was made. On opening the peritoneum a small amount of yellow serous fluid escaped and the cord immediately presented itself into the wound—it was pulsating. The appendix appeared normal. As the abdominal cavity was entered the fetus was found lying free. It was extracted and the cord was clamped and cut. The fetus cried spontaneously almost immediately. The placenta was noted adherent to many coils of intestine and covered the whole lower part of the abdomen. The tear in the amniotic sac was found. As the examining hand slowly moved to the left side and down into the pelvis a firm structure was found that had the feel, size and shape of a non-pregnant uterus. It was not visualized because of fear of separating the placenta. The cord was double ligated with chromic catgut and replaced into the abdomen which was closed in anatomic layers. The appendix was not removed. The patient was ambulatory after thirty-six hours and was discharged from the hospital on the eighth day. The fetus was placed in an incubator but did not require the use of oxygen. After twenty-four hours a fine polyethylene tube was passed into the stomach and the child was fed by gavage. After eleven days this tube was removed and the child was given a bottle with nipple and was able to feed itself satisfactorily. It has continued to thrive. Its birth weight was 2 pounds 11 ounces, and when last seen 3½ weeks after birth it weighed 3 pounds and 12 ounces.

COMMENTS

A case of ectopic pregnancy (abdominal) is reported in which the fetus was delivered alive and has continued to thrive. It is believed that the sharp pain this patient had at 4:15 A.M. on the morning of admission was probably due to rupture of the amniotic sac and the resultant irritation of the peritoneum by the amniotic fluid. Although at the time of operation the fetus was found lying free in the abdominal cavity, remnants of the torn amniotic sac were easily discernible.

L. A. BREWER has reported his experiences with surgery on the thoracic duct. Above the diaphragm, if the duct is injured with persistent leakage of chyle, he believes that simple ligation of the injured duct suffices. For abdominal fistula of the duct with large amounts of chylous material in the abdomen, he recommends anastomosis of the duct with the hemiazygos or azygos vein. (*Richard A. Leonardo, M.D.*)

Successful Treatment of Cardiac Herniation through a Surgical Rent in the Pericardium

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WITH the ever increasing number of cases of thoracic surgery which involve an excision of a portion of the pericardium for malignant disease of the lungs, the mediastinum or an intracardiac operation, it is important to draw attention to one of the more serious complications, cardiac herniation. Careful perusal of the English literature reveals only three references dealing with this complication, two with a successful result and a third in which the condition was unrecognized and death resulted. It is the purpose of this paper to present another case in which the outcome was successful.

CASE REPORT*

L. K., a fifty-three year old white man, underwent operation at St. Alexis Hospital for carcinoma of the left lung. (Fig. 1.) Because of extensive disease it was necessary to open the pericardium and to ligate the major vessels. A considerable portion of the pericardium had to be removed along with the lung in order to suture the left auricular openings of the superior and inferior pulmonary veins. The resultant defect was partially closed with one silk suture. At the end of the operation the patient's condition was good, his pulse and blood pressure were stable and his respirations were normal. On returning the patient to his room and immediately after moving him from a cart to his bed, it was noted that he suddenly went into shock with no radial pulsations or blood pressure. Further examination revealed that he had normal respirations and normal femoral arterial pulsations. Five hundred cubic centimeters of blood given rapidly intravenously under pressure did not improve his condition. Despite the fact that nasal oxygen was being administered the patient became cyanotic from the nipple line upward and difficulty in breathing developed. He was immediately intubated and positive pressure oxygen was given. However, the cyanosis of the upper portion of the body deepened and the femoral arterial pulsations became imperceptible. A large needle with an attached syringe was inserted into the chest and 100 cc. of bloody fluid was withdrawn. At this time, with a working diagnosis of massive intrathoracic hemorrhage, it was decided to reopen the left chest. Because of the graveness of the situation no attempt was made to move the patient to surgery, but rather the chest was reopened through the previous incision under unsterile precautions. Inspection of the left pleural cavity revealed that the heart was herniated out of the pericardial sac and was lying free in the left chest. It was beating feebly due to torsion of the great vessels. Hemostats were applied to the anterior and posterior margins of the pericardial opening and the heart was gently replaced. Following cardiac massage, the heart quickly regained its normal tone and color and a regular sinus rhythm was restored. The cyanosis immediately disappeared and his pulse and blood pressure returned to normal. On inspection of the pericardial rent it became apparent that when the patient was moved from the cart to his bed, sudden pressure had caused a dehiscence of the pericardial suture with tearing of the pericardium down to the apex. The rent in the pericardium was closed with four interrupted black silk sutures and the chest was closed in layers, leaving a thoractotomy tube in place. The patient was awake within a few minutes and except for a small subcutaneous wound infection made an uneventful and complete recovery. (Fig. 2.)

* Kindly referred by Dr. Leonard Backiel, Cleveland, Ohio.

COMMENTS

Herniation of the heart through a surgical rent in the pericardium is a serious complication constituting a surgical emergency. Since the primary defect is mechanical it is completely reducible provided the diagnosis is made and

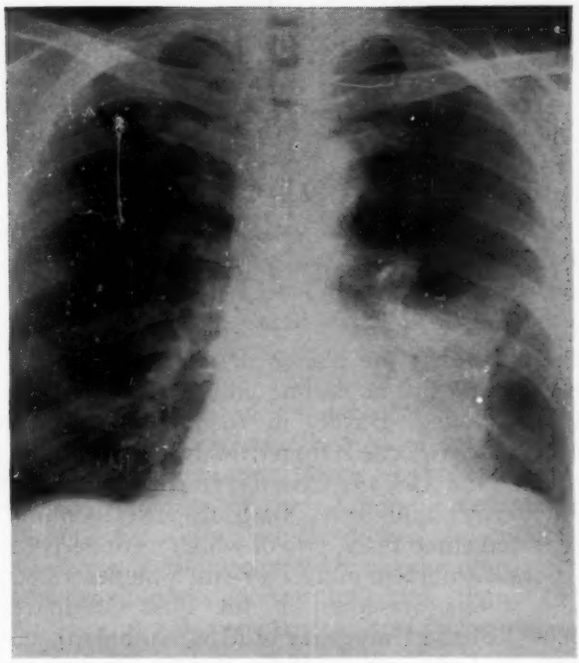

FIG. 1. Roentgenogram depicting the carcinoma of the lingular segment of the left lung.

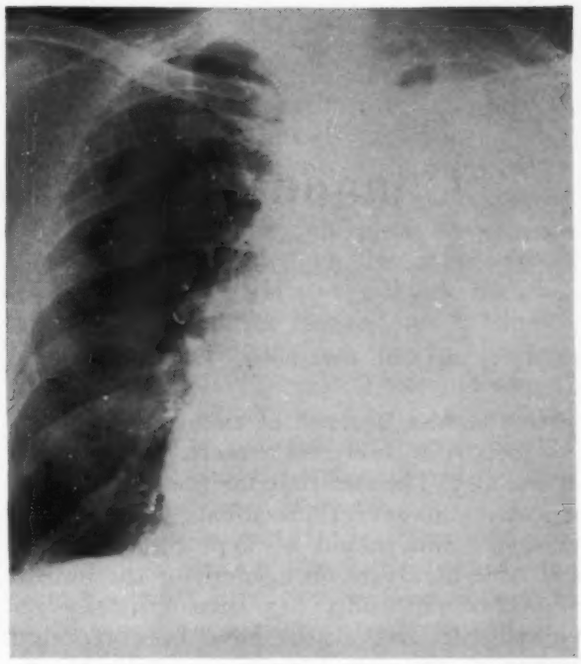

FIG. 2. Roentgenogram taken at the time of discharge from hospital.

treatment instituted in time. It occurs almost always within a few minutes of the termination of the procedure on the chest, at the time when the patient is first being turned or moved about, and so is of interest to both the anesthetist and surgeon.

The salient signs of cyanosis, unobtainable pulse and blood pressure, associated with normal respirations are a constant finding in all reported cases. The diagnosis is further strengthened if there is no mediastinal shift and only scant bloody return on tapping the involved pleural cavity. The case presented herein would appear to be quite typical in almost all respects except that the kinking of the great vessels at the base of the heart was not immediately complete, resulting in the short interval when the circulation was maintained only to the lower half of the body.

The differential diagnosis of cardiac herniation includes massive intrathoracic hemorrhage and cardiac arrest, but since the first step in the treatment of either of these is opening the chest, the differential becomes of little practical importance.

The treatment of the pericardium itself to prevent recurrence of the herniation consists either of closing it adequately, as was possible in the case presented herein because of the increased rent made during the herniation, or by laying the anterior pericardium wide open. (Fig. 2.) It would seem reasonable that these same methods employed at the time of original surgery would constitute excellent prophylactic treatment against this serious complication.

SUMMARY

1. A case of cardiac herniation through a surgical rent in the pericardium is presented with reduction of the hernia and uneventful recovery.

2. Immediate postoperative cyanosis, unobtainable pulse and blood pressure with a clear respiratory tree should suggest the diagnosis.

Acknowledgment: We wish to thank the Anesthesia Department (Drs. Lee Campbell and G. Rancoli), the surgical nurses and Drs. Joseph Bitzan and Walter MacLean without whose excellent cooperation this resuscitation would not have been possible.

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Congenital Absence of the Appendix

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CONGENITAL absence of the vermiform appendix is rarely encountered at operation or autopsy. The fact that the finding has been reported on several occasions confirms its existence and should be kept in mind. The available literature on agenesis of the human vermiform appendix has been critically reviewed and sixty cases have been reported to date.

Morgagni, in 1719, was the first to report absence of the appendix. In 1762 Hunter reported a case in which, during an operation for hernia, a cecum without an appendix was found. In 1812, Meckel described a case in which no trace of an appendix could be found at autopsy although the cecum was normal.

Dorland, in 1925, collected thirty-seven cases from the literature comprising those found at operation and autopsy. In twelve of these cases laparotomy had been performed after a diagnosis of acute appendicitis was made, and a thorough search had failed to discover any trace of the appendix. After 1925, cases were reported by Maurer, Blair Bell and Jacob. In 1925 Bradley reported a case in which a diagnosis of acute appendicitis was made and laparotomy performed. Instead of the appendix only a small protrusion, 1 cm. in diameter and 3 mm. in height, was present at the place of union of the three taeniae.

Spivack, in 1931, reported two cases found at autopsy which then brought the total number to forty-six. In 1938, Rambo and Lasky reported two cases. The first, an eighteen year old white boy, was operated upon for acute appendicitis and was found to have a congenital absence of the cecum and appendix; enlarged mesenteric lymph glands were present. The second, a twenty-two year old white man, was also operated upon for acute appendicitis, but was found to have a congenital absence of the appendix; the glands in the mesentery of the cecum and ascending colon were enlarged and inflamed. Boyce, in 1949, reported the only case recorded from the Record Library of the New Orleans Charity Hospital between July, 1937 and July, 1946. In 1951 Collins reported three cases, two of which were derived from postmortem material (which he described in a thesis presented at the Post Graduate School of the University of Minnesota in 1932) and one which was discovered at operation. Since that time further reports have appeared in the literature to bring the total to sixty-one cases, including the one to be reported herein.

CASE REPORT

F. G., a ninety year old white man, was seen on August 8, 1953, with the complaint of acute pain in the abdomen. The patient had been in fairly good health up to the present onset of illness, except for blindness following surgery for glaucoma thirteen years previously. Pain started in the right lower quadrant of the abdomen about 10:30 A.M. and increased in severity. Late in the afternoon he experienced nausea but no vomiting. The abdomen was becoming extremely painful, distended and rigid on the right side. The patient stated that bowel movements were regular every day and were of regular consistency. There had been no symptoms of gastrointestinal disorder and his appetite had always been good. He had not lost weight in recent years and had no symptoms of genitourinary disturbances. Considering his age and blindness, he managed to get about without difficulty. He seemed to be fairly alert and cooperative, and responded to questions readily.

Examination disclosed a swelling on the right side of the abdomen. There was marked tenderness over McBurney's point and rigidity on the right side with elevation of a circumscribed mass. His temperature was 98.6°F.,

heart rate 76, and blood pressure 110/70. A reducible left indirect inguinal hernia measuring 3 by 4 cm. was noted. The patient had been aware of this for the past thirty years, but it had not troubled him. The patient was short and very slender, weighing about 90 pounds. When he was first seen, a diagnosis of acute surgical condition of the abdomen was made, although the specific underlying cause was not too certain. Acute appendicitis with peritonitis was primarily considered although it seemed a bit farfetched to have such a condition occur in a man of his age. Torsion of the omentum, acute perforation of an ileocecal malignancy (although no suggestive history of irregularity of the gastrointestinal tract could be ascertained), volvulus of the cecum and mesenteric thrombosis were also considered as possible diagnoses. Laboratory examination soon after admission revealed the following data: red blood cells, 3,850,000 per cu. mm.; white blood cells, 9,800 per cu. mm.; hemoglobin, 77 per cent, 12 gm. per cent. Urinalysis showed a trace of albumin, 30 to 40 red blood cells and no casts. Repeat urinalysis on August 10th showed a trace of albumin. The entire field was covered with white blood cells, but no red blood cells were seen. Immediate surgery was decided upon instead of delaying for further observation and study with x-rays. It seemed that if any relief of the acute condition could be obtained, especially in such an elderly man, it would have to be investigated as soon as possible before changes in the abdominal organs would become irreversible.

Under light pentothal® gas-oxygen-ether anesthesia, the abdomen was opened through a right pararectus incision in the paraumbilical region. The peritoneum was tense and when opened, free bloody fluid was present. The cecum was sought in the right gutter but was found to lie freely toward the midline, acutely distended to about 8 by 10 cm. and twisted on its long mesentery clockwise one and one half times along with the ascending colon. This was followed distally and was found to be free from attachment to the lateral wall up to the hepatic flexure. The twist in the cecum and ascending colon was relieved by rotating the colon counterclockwise. The fluids and gases were gently expressed from the cecum toward the transverse colon, collapsing the distended cecum which appeared viable, although thinned out, and acutely congested. The appendix was looked for at the end of the taeniae coli and about the junction of the ileocecal valve, but there seemed to be no trace of it. There was no indication of its atrophic former existence. It must have been congenitally absent along with the improper development of the right mesocolon embryologically. The ileum was inspected and found to be normal. There was no evidence of a Meckel's diverticulum. No attempt was made to fix the cecum to the lateral wall because of its friable or thinned out condition. The liver was normal in size and consistency, but the gallbladder was markedly distended and full of small stones. This was removed without difficulty, a Penrose drain was inserted to Morrison's pouch through a stab wound, and the wound was closed in layers.

The patient tolerated the operation well. Wangensteen suction was instituted and the patient was fed intravenously for two days, at which time he was allowed to get out of bed. He continued to do well and had a normal bowel movement on the fourth day. The clips were removed on the fifth day, and the patient was discharged from the hospital on August 15, 1953, just one week after admission and surgery. He remained well at home till June, 1955, when he died following a fall incurred by his blindness.

COMMENTS

Congenital absence of the appendix is a rare anomaly and should not be confused with absence due to atrophy, absorption or amputation subsequent to suppurative processes. Local pathologic processes were lacking in the case reported herein, and there was no diverticulum or tubercle demonstrable which might represent the appendix. Complete failure of the appendiceal anlage to differentiate results in primary congenital absence of the organ.

It is known from comparative anatomy that the appendix is congenitally lacking in almost all mammals except man. It is present in anthropomorphous apes and in some marsupials, while in some rodents it is represented by a mass of lymphatic follicles in the distal portion of the cecum which is derived from the distal branch of the primitive intestinal loop. There, at a short distance from the implantation of the vitelline duct, very early at the free margin a small dilatation appears in the form of a diverticulum which represents the anlage of the cecal apparatus. This diverticulum

becomes elongated and gradually assumes a uniform caliber, later assuming the shape of a funnel, the apex of which corresponds to the appendix. Growth becomes unequal and the lateral wall grows more than the medial one, so that the root of the appendix is displaced medially and backward from the most declivous point. Lacking thinning in the form of a funnel concerning the lower cecal segment determines subsequent total absence of the appendix. This arrest of the embryonal development of the cecum is dealt with, leading to total absence or to formation of a very short appendix which is hypoplastic or rudimentary. Appendices the size of a lentil have been reported. The congenital absence of the appendix and the redundant mesentery of the cecum and ascending colon, with absence of fixation to the lateral abdominal wall to produce a volvulus of the cecum, may have had some relationship in this case.

SUMMARY

1. Congenital absence of the vermiform appendix is a medical rarity that has been reported in the literature and found in clinical conditions simulating acute appendicitis or at autopsy in about sixty cases.

2. Complete failure of the appendiceal anlage to differentiate is believed to result in a primary congenital absence of the appendix.

3. An additional case is reported in a ninety year old man whose acute surgical condition was found to be volvulus of the cecum.

4. The congenital absence of the appendix and the redundant mesentery of the cecum and ascending colon that tended to produce a volvulus of the cecum may have had some relationship in this case.

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NEW INSTRUMENT

A New Binocular Pharyngolaryngoscope

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WHILE it is common knowledge that a completely undistorted binocular (stereoscopic) view of the hypopharynx and larynx can be obtained only by mirror examination, nevertheless, direct endoscopy is necessary to obtain a full view of the vocal cords in certain patients in whom the epiglottis tends to remain tipped backward so as to obscure the glottis, even though the tongue is pulled on rather forcibly. Although instrumentation (biopsy) of the pharynx can often be guided and controlled by indirect or mirror endoscopy and a curved instrument, manipulations at the glottic level almost always require direct laryngoscopy.

Heretofore the classic instruments for direct laryngoscopy (or pharyngoscopy) have consisted of narrow tubes of rather small and uniform caliber, permitting only monocular vision. It is obviously impossible to employ tubes of uniform caliber sufficiently large to permit binocular vision. Suspension laryn-

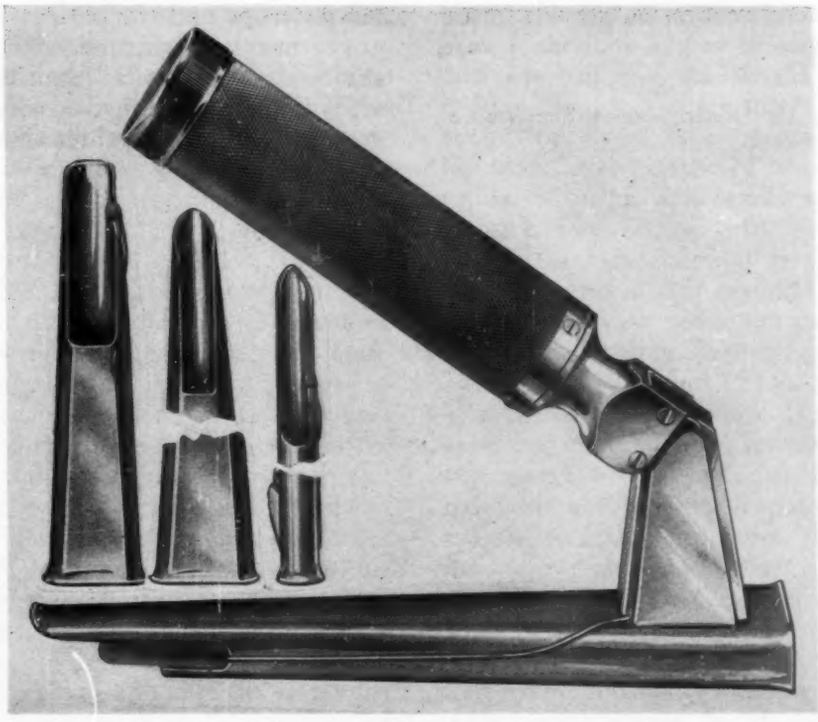

FIG. 1.

goscopy is not the complete answer since the procedure is complicated, troublesome and somewhat risky.

The new binocular endoscopes described herein* are tapered so as to give a binocular (stereoscopic) view of the hypopharynx and

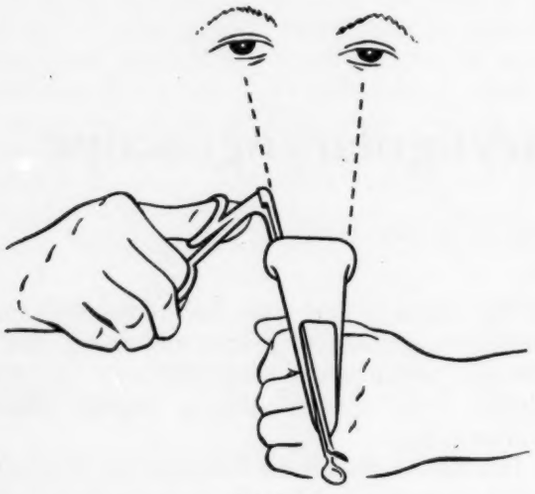

FIG. 2.

larynx. These tapered tubes permit the introduction of a biopsy forceps or other instruments along one of the side walls without interfering with or obscuring the view, as is the case with monocular endoscopes. To provide for the various requirements in the individual case, three separate blades are supplied, each of which fits one universal handle: (1) A binocular tapered blade, with a broad, flat tip, for examination of the base of the tongue, pharyngeal walls and extrinsic larynx. (2) A binocular tapered blade, with a pointed tip, for inspection of the interior of the larynx and the glottis. This binocular blade is particularly suitable for direct laryngoscopy under general anesthesia since it permits accurate instrumentation under binocular vision. Its use is not always practical with topical anesthesia. (3) A monocular nontapered blade of generous although uniform caliber for use in uncooperative patients or in difficult cases in which the wider tapered blades would not be practical. The handle is set at an angle to the blade itself, a position which is a compromise between that parallel to the blade, as in the Jackson instrument, and that set at right angles, as in many modern laryngopharyngoscopes. This compromise has many advantages of each of the other positions, with a minimal degree of their disadvantages. (Figs. 1 and 2.)

The source of the electric current may be from the standard type of flashlight batteries contained in the handle or from a special, fixed transformer attachment for connection with the standard 110–120 voltage house current. The transformer insures that only a correct and safe amount of voltage reaches the laryngoscope and the patient. The light bulbs are of heavier construction and are much less subject to "burn-outs" than the small (wheat seed) bulbs used with the older type of laryngoscopes, and furnish three times more light.

* Distributed by the United Surgical Supplies Co., Mamaroneck, New York.

BOOK REVIEWS

Practical Office Gynecology. By Albert Decker, M.D., F.A.C.S., Diplomate, American Board of Obstetrics and Gynecology, and Wayne H. Decker, M.D., Diplomate, American Board of Obstetrics and Gynecology. (One of a series of monographs, edited by Claude E. Heaton, M.D.). Cloth, 388 pages, 103 illustrations, 19 in color, with bibliography and index. Philadelphia, 1956. F. A. Davis Co. *Price* $10.50.

The Deckers, father and son, have written a noteworthy book on office gynecology. It is called a practical text, which is true in every sense of the word. The writing is to the point; one is impressed with the lack of "padding." Unlike many scientific texts the writing in this work has a style which approaches professional authorship. The colored illustrations are excellent.

The field of office gynecology is completely covered. We found certain chapters most valuable, namely, Hysterosalpingography, Cul-de-sac Puncture, Culdoscopy (although Dr. Albert Decker, who designed or invented the culdoscope, describes the technic of its use, he stresses it is not an office procedure), Abnormal Genital Tract Bleeding, Infertility, Psychosomatic Gynecology, Pelvic Tumors, Indications for Gynecologic Surgery (every young doctor should memorize this) and Cancer Detection in Women.

There have been other books on this subject (we have in our library a tome with the same title which was published just ten years ago). However, during the past decade a new era of medicine has reached mature proportions, and gynecology is no exception to the general rule. Therefore it is no exaggeration to state this monograph is up-to-the-minute and modern.

The mature, finished gynecologist might feel this work is not for him. However, the young practitioner, whether a third or fourth year medical student, intern or resident, or the "family doctor" in general practice, will find this book invaluable as an aid in his every day work.

The Principles and Art of Plastic Surgery. By Sir Harold Gillies, C.B.E., F.R.C.S. and D. Ralph Millard, Jr., M.D., Diplomate, American Board Plastic Surgery, Assistant Clinical Professor, University of Miami School of Medicine. Chapter on Anaesthesia by Jerome Magiel, C.V.O., F.F.H.R.C.S. Foreword by Jerome Pierce Webster, M.D., Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University. Two volumes, boxed. 652 pages, profusely illustrated, many in color, with biographical data and an index. Boston, 1957. Little, Brown & Co. *Price* $35.00.

We believe a careful perusal and study of this work will be rewarding to all interested in plastic surgery. The student, intern or resident who aims one day to confine his future career to this surgical field should own these volumes. One might rightly conclude this is a solid, authoritative and excellent writing.

It might be well to give an outline of the contents of the two volumes. The text of Volume One begins with The First Ace—Cinderella Surgery, which deals with World War I, principles, anesthesia, technical tips, and skin and inlay grafting. The next heading is Flap Happy. We are instructed in rotation and direct flaps, tube pedicles, lymphedema, forehead flaps, pigmented nevis, hemangioma, radiation burns, and candor and ear making. Volume Two begins with Harelip and Cleft Palate, and is followed by Rag Bag, and Genitalia. After this comes Private Practice which deals with reduction and aesthetic surgery. The following chapters, under the heading Trauma in War and Peace, deal with World War II and Rooksdown House, burns, an aspect of hand surgery, lip trauma, surgery of the mandible and maxilla, fractures of the mandible and maxilla, fractures of the malar-zygomatic compound, nasal fractures, frontal defects, eyelids and sockets, facial paralysis and cross-grafting. The second volume ends with Final Scene, A Day in the Clinic, and Epilogue.

We estimate there are at least 200 or more half tones, and many illustrations are in color. Those illustrations in black and white are

uniformly excellent; those in color are among the best we have seen in recent times. The text is to the point and well written. Those interested in this subject will find a text that satisfies. These volumes deserve wide distribution.

Fluid Balance Handbook for Practitioners. By William D. Snively, Jr., M.D. and Michael J. Sweeney, M.D. 308 pages, illustrated, with references for additional reading and an index. Springfield, Ill., 1956. Charles C Thomas. *Price* $6.75.

Although much has been written on fluid balance, it is the general impression that many physicians have only a hazy and at times an inaccurate conception of the subject. Therefore this monograph is timely and fills a void. The authors say their goal in preparing this handbook has been to provide the practicing physician with an inviting, clear and usable text on *clinical* fluid balance. The writers have accomplished their purpose.

The book is divided into six parts: (1) Foundation Facts; (2) Clinical Diagnosis; (3) Therapy: Changes in Properties of Extracellular Fluid; (4) Therapy: Changes in Position of Extracellular Fluid; (5) Therapy: Changes in Nutritional Status of Body; (6) Therapy: Routes of Administration; (7) Down to Cases. There is an Epilogue.

It is true that the authors have "spoken the language of the clinician, not of the biochemist," and that they have "viewed the subject from the standpoint of the needs of the practicing physician." We are informed that the authors submitted the preliminary manuscript to a panel of forty practitioners, academic physicians and technically trained laymen for helpful criticism and suggestions.

All in all this monograph is an up-to-date "working manual."

Surgery for General Practice. By Victor Richard, M.D., Professor of Surgery, Chairman of the Department of Surgery, Stanford University School of Medicine, San Francisco, Calif. 947 pages, 476 illustrations, with an index. St. Louis, 1956. C. V. Mosby Co. *Price* $17.50.

Dr. Richards and his seven contributors have written an excellent book. The contributors are Drs. Francis J. Cox, Roy B. Cohn, Frederick Howard, Russell R. Klein, Carleton H. Mathewson, Robert McNaught and Charles McLennan, all specialists in their chosen surgical fields. We read: "This book will be of value and of real usefulness to the average physician caring for the majority of surgical problems in the everyday practice of medicine, whether he be general practitioner, student, internist or surgical specialist. Address is specially made to those who are in want not of additional facts but of a better organization of facts already available . . . There is a growing tendency for the average physician to be unable to organize for his use the existing knowledge of the day, and this book is an effort to organize for him surgical facts in a usable practical array."

The background for this book grew out of over ten years' experience in the Stanford University Surgical Outpatient Clinics.

In between the first chapter on Local Anesthesia and the final one on Fractures are forty-one chapters full of significant, up-to-the-minute information. One can read through this book and experience a mental satisfaction when the reading is finished.

Mrs. Jean Dunlap has done a workmanlike job in making the sketches, drawings and illustrations clear and simple.

Although the finished surgeon of long and wide experience may conclude he does not need this work, it is the reviewer's opinion that the intern, the surgical resident and the general practitioner will do well to add this book to his library. He should read it from cover to cover, and from time to time reread many parts. He is sure to add to the sum total of his knowledge and thus become a better practitioner.

The Merck Manual of Diagnosis and Therapy. Editorial Board: Charles E. Lyght, M.D., Editor, Wm. P. Boger, M.D., Geo. A. Carden, M.D., August Gibson, M.D. and Dickinson W. Richards, M.D. Ninth edition. 1,888 pages. Rahway, N. J., 1956. Merck & Co., Inc. *Price* $6.75 regular edition, $9.00 de luxe edition.

Authors and consultants for this edition include more than 100 leading clinicians in the United States, Canada and abroad. With much of its contents revised, the new edition of The Merck Manual carries more illustrative material than the previous edition. There are 378 principal chapters on the diagnosis and therapy of diseases. There are twenty main sections, each thumb-indexed, covering specific fields of practice.

More than 1,600 prescriptions are included, embodying the most up-to-date medicinal advances. Special procedures described include Bedside, Clinical, Preoperative, Postoperative and Office Laboratory.

Diagnosis and therapy are given primary emphasis, but pertinent physiologic, pathologic and etiologic features are included. Up-to-the-minute coverage has been given to adrenocortical and related therapy, "tranquilizing" drugs and antituberculosis compounds, as well as to agammaglobulinemia, kwashiorkor, and mucoviscidosis.

Discussions of modern advances in the fields of antibiotics, nutrition, immunization, Rh factor, gamma globulin, and pre- and postnatal care are extensive and bring together current accepted developments on these subjects.

A wealth of readily applicable advice is set forth on such vital subjects as medical emergencies, burns, poisoning and shock. Reference values and conversion data are included in addition to an index which is liberally cross-titled and substantially expanded over previous editions.

Diseases of the Breast. By C. D. Haagensen, M.D., Professor of Clinical Surgery, The College of Physicians and Surgeons, Columbia University, Director of Surgery. The Francis Delafield Hospital, Columbia-Presbyterian Medical Center, New York, N. Y. 751 pages, illustrated with 404 figures and 25 charts, with an index of authors and subjects. Philadelphia, 1956. W. B. Saunders Co.

An outstanding book on the breast by a surgeon who writes he has " . . . tried to present a synthesis of what I have learned during twenty-five years of specialized concentration on diseases of the breast . . . The book is based not only upon my personal experience, but upon all the data concerning diseases of the breast which have accumulated in our records (Columbia-Presbyterian Medical Center) during the forty year period 1915 to 1955."

When one realizes the leading, nationally known scientists under whom and with whom Dr. Haagensen has worked—Ewing, Auchincloss, Arthur Stout, Allen Whipple, Lattes, Maurice Lenz—this work naturally reflects the labors of many experts in this field.

The scope of the book is broad in every sense of the word. It is a complete book; we cannot discover anything which has been omitted or passed over with but a scanty paragraph. It is a reference book as well as a textbook. There are thirty-one chapters. As with many monographs, the opening chapter deals with the anatomy, then the physiology, and then goes on to discuss hypertrophy of the breast, the detection of breast disease and methods of diagnosis of breast disease. We cannot name the chapter headings in their order. Suffice to affirm that the various disorders of the breast, their diagnosis and treatment are covered in detail. Especially good and profitable reading are the chapters on The Natural History of Breast Carcinoma, The Symptoms of Mammary Carcinoma, The Papillary Type of Mammary Carcinoma, Inflammatory Carcinoma, The Choice of Treatment for Breast Carcinoma and The Surgical Treatment of Mammary Carcinoma. One chapter is devoted to cancer of the male breast. The discussions of the administration of hormones, surgical and x-ray castration for carcinoma of the female breast, and orchiectomy for malignancy of the male breast, and adrenalectomy and hypophysectomy leave nothing to be desired. The illustrations are above the average.

It is an excellent and valuable book which the general practitioner will find a great aid in his everyday work (he will give the breast attention and learn how to suspect or note trouble). The intern, surgical resident and the young surgeon should mark this book in their "must" column.

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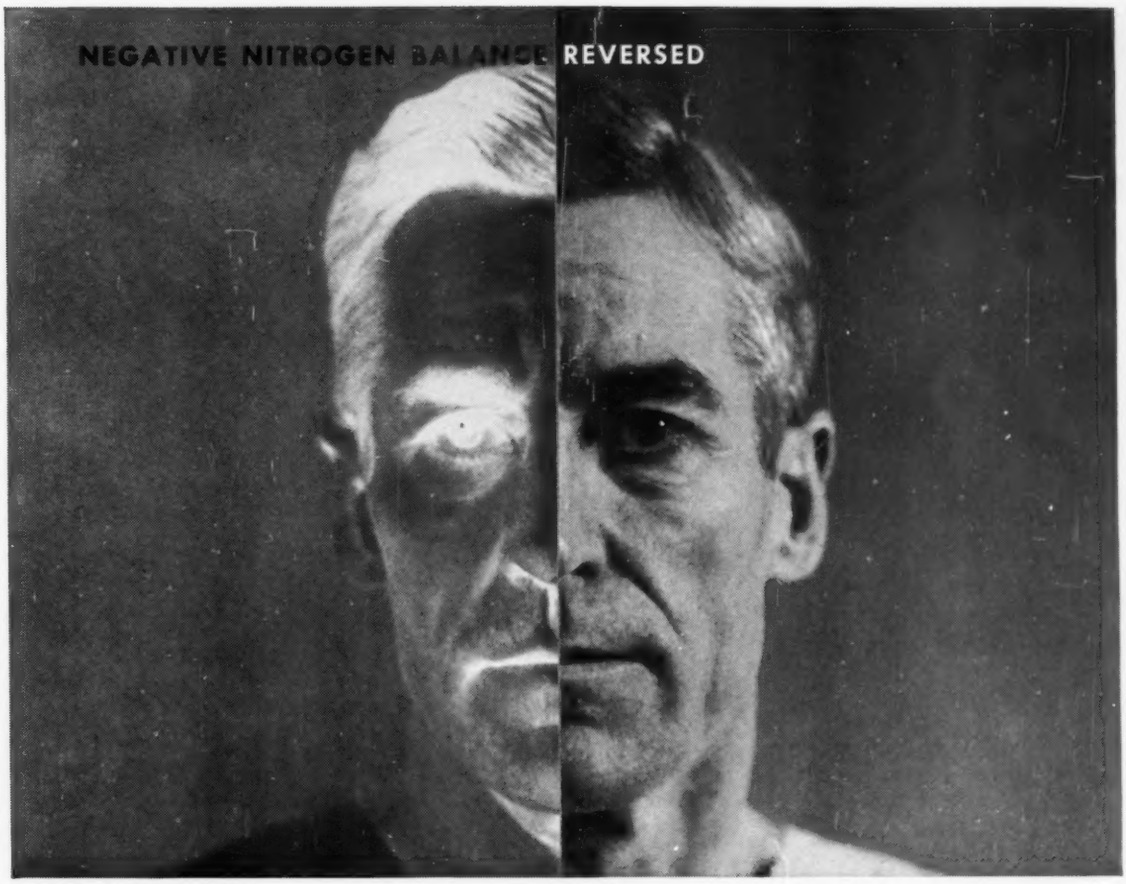

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1. Axelrod, A. E.; Beaton, J. R.; Cannon, P. R., and others: Symposium on Protein Metabolism, New York, The National Vitamin Foundation, Incorporated, (March) 1954, p. 100.

2. Proceedings of a Conference on the Clinical Use of Anabolic Agents, Chicago, Illinois, G. D. Searle & Co., April 9, 1956, pp. 32-35.

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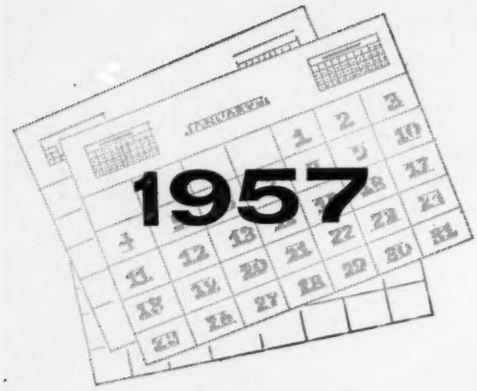

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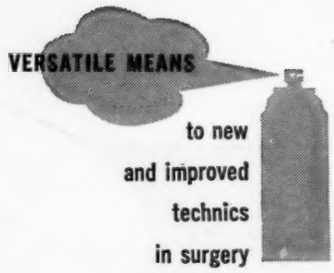

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1. Bacala, J.C.: The Use of the Systemic Hemostat, Carbazochrome Salicylate, West. J. Surg. *64*:88 (Jan., 1956)

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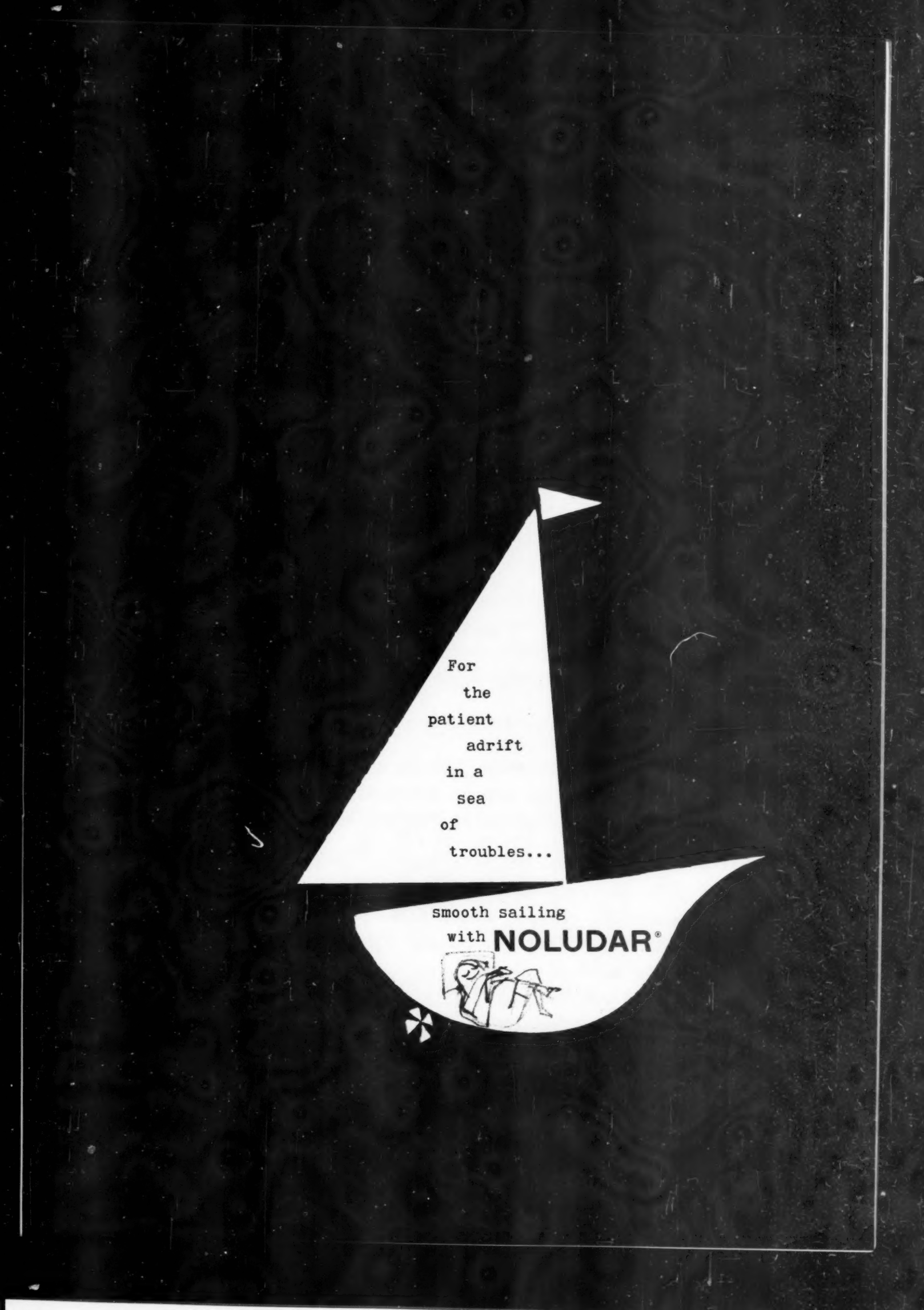
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2. Susinno, A. M., and Verdon, R. E.: J.A.M.A. *154*:239, 1954.

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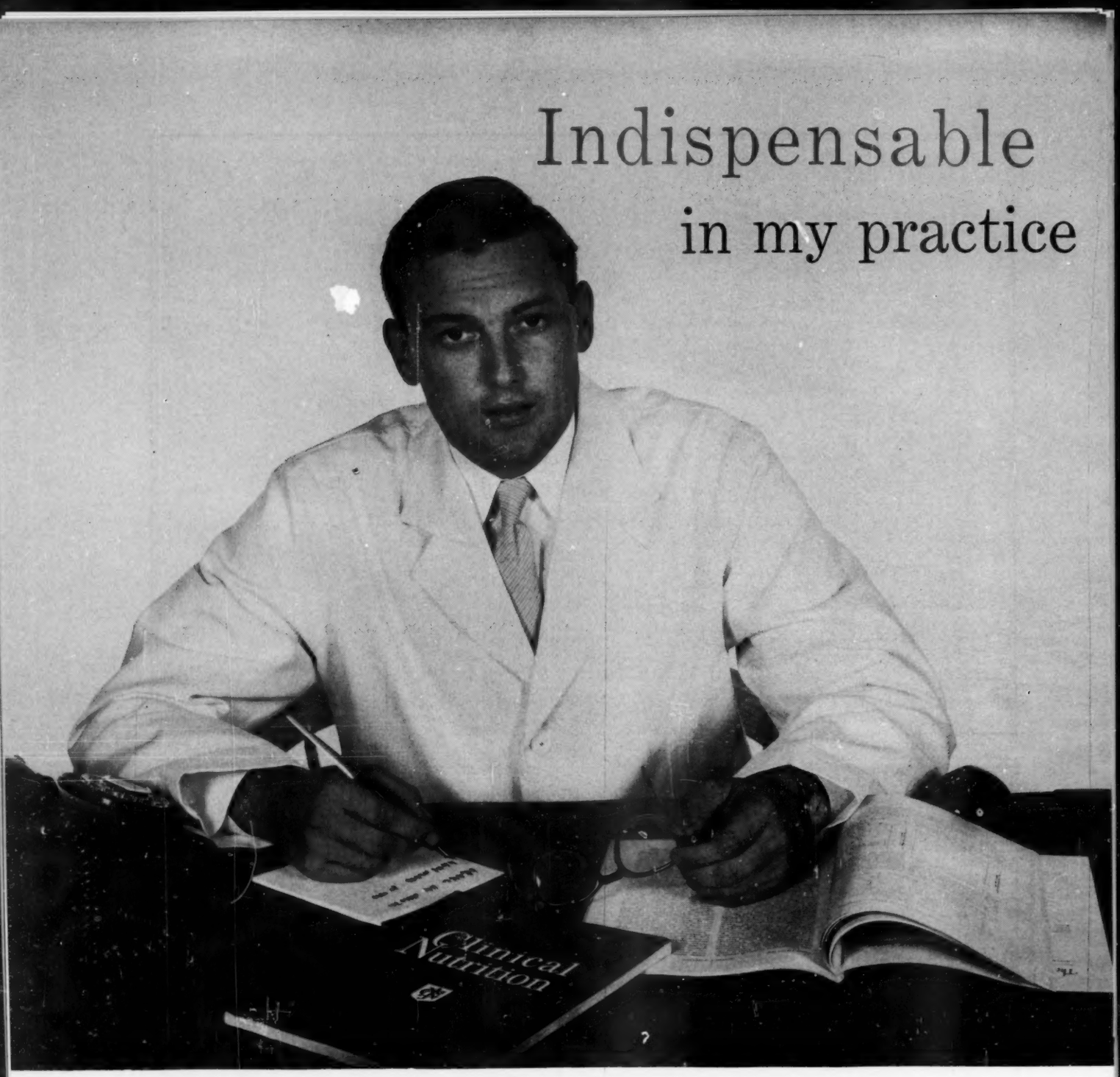

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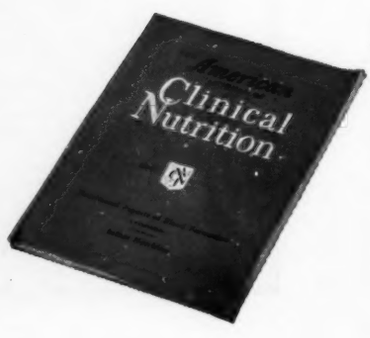

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1. Elman, R., and Cannon, P. R., in Clinical Nutrition, Jolliffe, N., et al., eds., New York, Paul B. Hoeber, Inc., 1950, p. 183.

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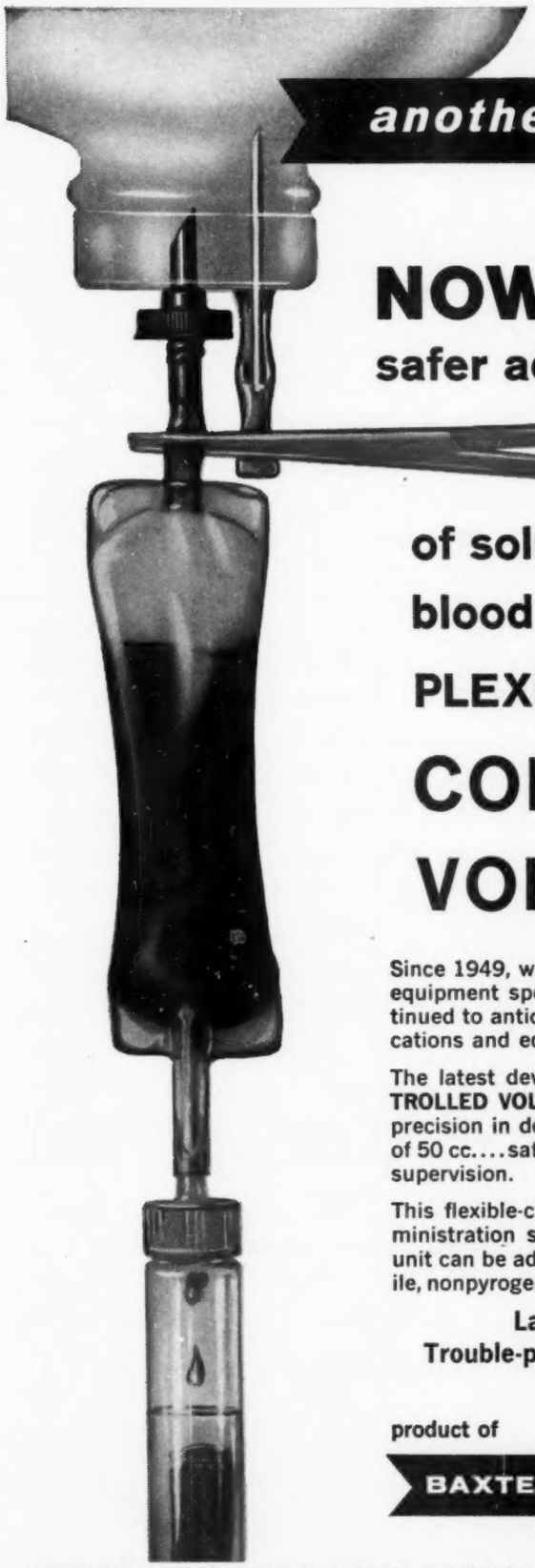

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The sharp increase in nutritional requirements during adolescence is ascribed to the rapid growth, restless activity, high basal metabolism, and increased rate of organ development during this period.[1, 2] Nutrient needs during adolescence are higher than at any other period of life[3] except for pregnancy and lactation.

In order to satisfy these extremely high nutritional requirements, "protective" foods supplying liberal amounts of protein, vitamins, and minerals should predominate in adolescent diets.[3] Such foods include meat, poultry, fish, milk, eggs, vegetables and fruits, and whole-grain or enriched cereals and enriched bread. Accessory foods commonly eaten by adolescents to satisfy emotional needs may provide energy, but are commonly responsible for obesity and should not take the place of the "protective" foods.

Meat contributes much toward making the daily meals of adolescents appetizing, ample, and satisfying as well as adequate in protein, B vitamins, iron, phosphorus, potassium, and magnesium. Its complete protein functions in all physiologic mechanisms utilizing protein—tissue growth and replacement, fabrication of enzymes, hormones, and antibodies, and maintenance of the body's fluid balance. Its B vitamins and minerals take part in many processes of intermediate metabolism important in body development.

1. Toverud, K. U.; Stearns, G., and Macy, I. G.: Maternal Nutrition and Child Health. An Interpretative Review, Washington, D.C., National Research Council, National Academy of Sciences, Bull. No. 123, 1950, p. 115.
2. Proudfit, F. T., and Robinson, C. H.: Nutrition and Diet Therapy, ed. 11, New York, The Macmillan Company, 1955, p. 271.
3. Martin, E. A.: Roberts' Nutrition Work with Children, Chicago, The University of Chicago Press, 1954, pp. 231-236.

The nutritional statements made in this advertisement have been reviewed by the Council on Foods and Nutrition of the American Medical Association and found consistent with current authoritative medical opinion.

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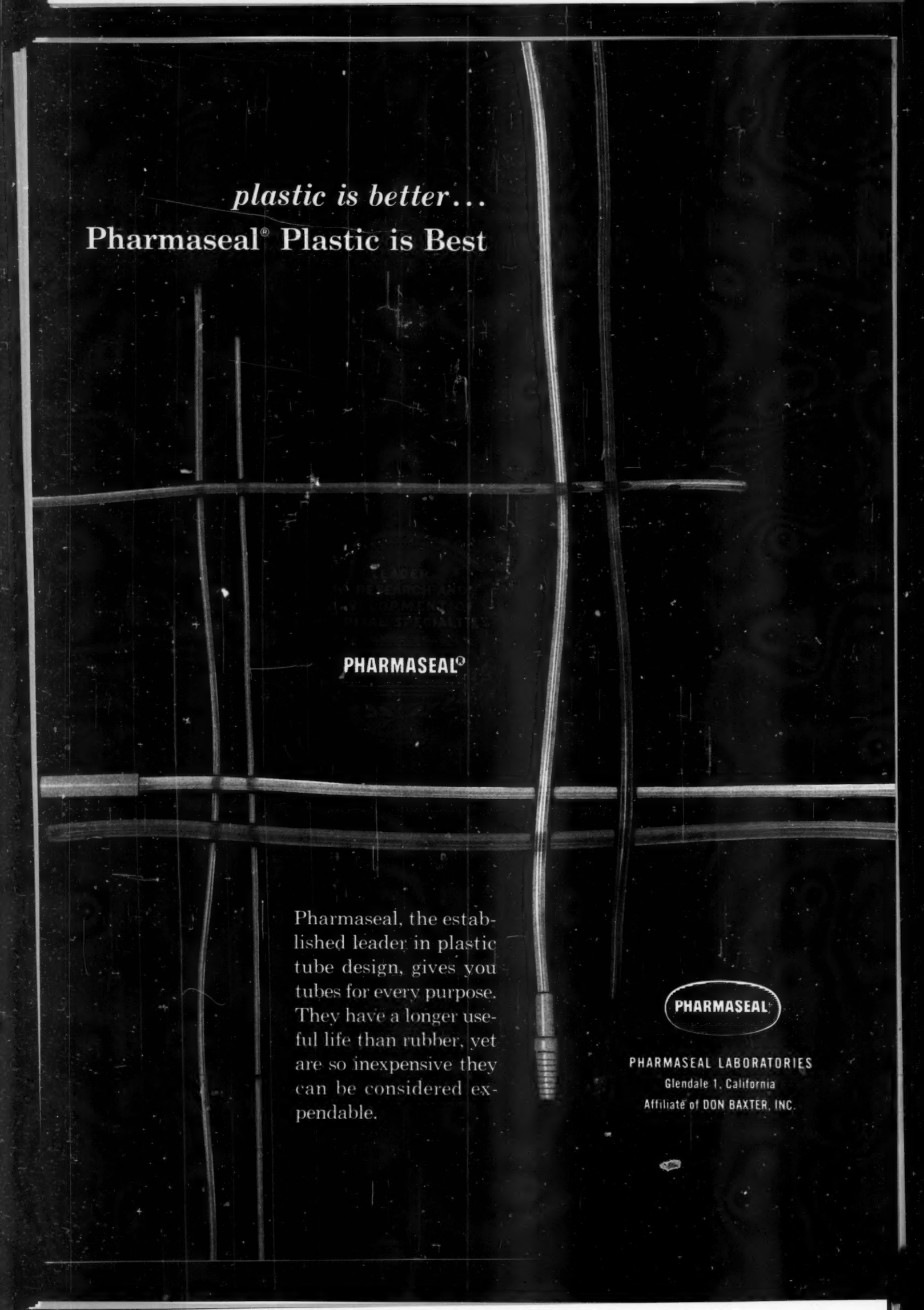
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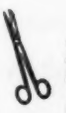

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NOTES AND QUOTES

THE AMERICAN CYANAMID COMPANY has announced that grants totalling $235,612 have been made by the Company to thirteen outstanding medical school teachers and researchers for its 1957 Lederle Medical Faculty Awards. In making the announcement, K. C. Towe, President, said that these awards, which were initiated by Cyanamid in 1954, were believed to be the first of their kind in the field.

"The basic purpose of the grants is to help assure to schools and universities adequate funds to encourage medical men of stature to continue in teaching and research posts," explained Mr. Towe. "The awards are intended to supplement existing salaries or to aid schools in filling positions with medical men of highest calibre.

"To help insure the health of future generations of Americans, we feel that in no other way could our Company make more significant contributions to our great medical teaching institutions."

The tranquilizing drug miltown® is being offered in a new dosage form, a sugar-coated 200 mg. tablet, by Wallace Laboratories, New Brunswick, New Jersey. The drug had previously been available only as an uncoated 400 mg. tablet.

Miltown is a muscle relaxant as well as a tranquilizer, and is used in the treatment of skeletal muscle spasm in cerebral palsy, rheumatic conditions and various neurologic disorders. It is also used in treating petit mal epilepsy.

THE GEORGE P. PILLING AND SON COMPANY has recently introduced a new multi-purpose bronchoscope, the "pentascope," designed for Dr. A. Albert Carabelli of Trenton, New Jersey. The pentascope can be used for examination, catheterization, biopsy, fulguration and foreign body removal in all five lobes of the lung. It consists of a tube, light carrier and sheath, deflector and operating channel insert, and accessory collars and adapters.

The pentascope accepts Jackson telescopes so that the bronchoscopist can work under direct vision in areas heretofore practically inaccessible, such as the upper lobes. The bronchoscope measures 6 mm. for the inside lumen, 8 mm. for the diameter and is 40 cm. long. The pentascope also features a distal inspection port as well as oxygen, suction and anti-fog features.

The Carabelli pentascope can be seen at Pilling's New York showroom at 4 West 56th Street, New York, as well as in the Philadelphia headquarters, or on display at many surgical conventions and meetings.

Hospital and medical care payments, to help cover the cost of treatment and physicians' services, amounted to $2.1 billion in 1956, the Health Insurance Institute reported recently. This figure, the Institute stated, includes replacement of income lost through sickness or disability. A survey conducted among the country's insurance companies revealed that reimbursements through group insurance plans in force during 1956 totaled $1.5 billion, or 20.9 per cent over 1955, while payments through individual policies totaled $601 million, a gain of 12.8 per cent, for an over-all increase of 18.5 per cent in benefit payments over 1955.

Payments to policyholders covered under hospital expense insurance, for inhospital services, amounted to over $855 million, with $629 million paid under group policies and $226 million paid by insurance companies to individual policyholders.

Reimbursements to help cover the cost of surgeons' fees amounted to $346 million, with $273 million received by holders of policies under group plans, and $73 million going to persons covered by individual insurance policies.

A total of $58 million was paid to persons under regular medical expense contracts, for non-surgical medical care and treatment, $47 million through group policies and $11 million to individual policyholders.

THERE is no evidence that the tranquilizer drugs are habit-forming or addictive, U.S. Narcotics Commissioner Harry J. Anslinger told a Congressional subcommittee recently. On the contrary, Dr. Anslinger indicated, the tranquilizers have proven to be "of terrific value" in relation to the U.S. mental health problem.

In reply to a question by Rep. Gordon Canfield (N.J.), Dr. Anslinger pointed out that the Committee on Narcotics of the National Research Council has been considering

CONTINUED ON PAGE 48

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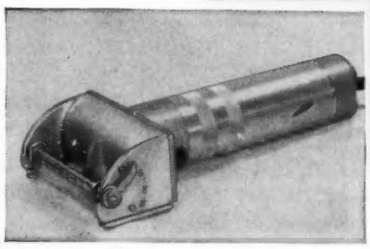

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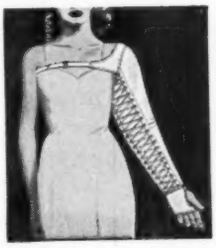

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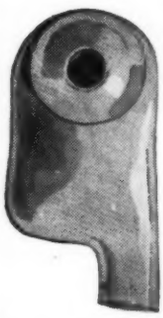

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the possible danger of drug addiction from use of the tranquilizer drugs. The key evidence of addiction, Dr. Anslinger said, is the drug addict himself, "and you do not see a drug addict touching those (tranquilizer) drugs. They do not even try them."

Indicating the need for careful medical supervision of patients taking drugs, Dr. Anslinger indicated that heavy "non-therapeutic" doses of any drug, such as the barbiturates, may lead to addiction.

"But we have seen nothing in this orbit" in connection with the tranquilizers, he said, "and I am thankful, because I think these drugs, in relation to the mental-health problem in this country, are of terrific value."

The tranquilizer drugs, or ataractics, have recently found broad application in the treatment of both hospitalized mental patients and non-psychotics suffering from anxiety, tension and related symptoms. Reserpine and chlorpromazine generally are used in mental hospitals, while tranquilizers such as meprobamate are prescribed for less severe cases of anxiety and tension.

Applications for certification for the American Board of Obstetrics and Gynecology, new and reopened, for the 1958 Part I examinations are now being accepted. All candidates are urged to make such application at the earliest possible date. Deadline date for receipt of applications is September 1, 1957. No applications can be accepted after that date.

Candidates for admission to the examinations are required to submit with their application a typewritten list of all patients admitted to the hospitals where they practice for the year preceding their application or the year prior to their request for reopening of their application. This information is to be attested to by the Record Librarian of the hospital or hospitals where the patients are admitted, and submitted on paper 8½ by 11 inches. Necessary detail to be contained in the list of admissions is outlined in the Bulletin and must be followed closely. Current Bulletins outlining present requirements may be obtained by writing to Dr. Robert L. Faulkner, American Board of Obstetrics and Gynecology, 2105 Adelbert Road, Cleveland 6, Ohio.

SPECIAL stainless steel connectors for artificial heart and lung shunt appliances are now available through E. Weck & Company, Brooklyn, New York. The inner lumen must be precisely tapered so that blood flowing from one tube to another via the connectors will receive no cell damage. All types and sizes are available on order.

Cancer-producing chemicals called carcinogens may constitute a mounting danger to everyone from the city librarian to an Olympic weightlifter, but only by further research can scientists learn the final answer.

Professor R. K. Boutwell of the University of Wisconsin's McArdle Memorial Laboratory points out that these carcinogens may be contained in many common everyday substances—from the smoke of cigarettes and polluted air to sunlight and possibly certain food additives.

However, he is quick to point out that not nearly enough is known about common carcinogens to be able to name them all with certainty.

Miners and workers in certain industries are the most likely persons to come in contact with carcinogens, especially if they work with arsenic, chromate, nickel, asbestos and other "dusty" materials.

"The dust from these substances gets into the lungs and often causes lung cancer," Professor Boutwell emphasizes. "Some of these materials can also produce cancer in other parts of the body—on the skin or in the bones."

The danger, however, is not limited to these workers.

"Whenever you have incomplete combustion, tarry residues naturally result," he says. "These residues have often been found to be carcinogenic in laboratory animals."

THE average length of life among the industrial policyholders of the Metropolitan Life Insurance Company rose to a new high of 70.2 years in 1956. This is slightly above the figure for 1955 and represents an increase of twenty-four years since 1909.

For nearly two generations the average length of life has been increasing more rapidly among American wage earners and their families than for the general population of the

CONTINUED ON PAGE 50

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NOTES AND QUOTES

United States. In 1909 the average lifetime of the Metropolitan's industrial policyholders was about six years less than that for the population as a whole. Since then, the disparity has been narrowing; at present the two groups are on a par.

Undergraduate scholarships worth $50,000 have been established by The Upjohn Company, Kalamazoo, Michigan, for the 1957–1958 school year.

Eight four-year scholarships will be given through the National Merit Scholarship Corporation, six of them to students who plan to major in pharmacy, engineering, pre-medicine, or any of the chemical or biological sciences. The other two may be in any field.

National Merit Scholarship Corporation is a non-profit organization which administers a nation-wide scholarship system. Selection of recipients is handled entirely outside of The Upjohn Company through a series of tests and interviews, so winners represent the nation's most gifted students. Each donation is matched by the Corporation from its endowment fund.

Scholarship support on a local basis is also provided in the new Upjohn program. A sum of $6,000 goes to Kalamazoo College: $2,000 for the general college fund and $4,000 to talented high school graduates in the area. State-supported Western Michigan College receives $4,000 for scholarships. Designated for science majors, the scholarships will be known as Upjohn Science Scholarships.

THE use by surgeons of hypothermia, or "refrigerated sleep," to suspend the work of the heart for repairs may be safer in the future with the application of new research findings by Public Health Service scientists.

Drs. Thomas A. Lombardo, Leo R. Radigan and Andrew G. Morrow of the U. S. Public Health Service's National Heart Institute of the National Institutes of Health at Bethesda, Maryland, have found in animal experiments that the threat of heart muscle failure in such operations can be greatly reduced by injections of strophanthidin, a digitalis-like "heart tonic" from the seeds of the African Strophanthus shrub. A description of the technic appeared in the January issue of *Circulation*.

The use of hypothermia permits many otherwise impossible heart operations because, by lowering body temperature 12 degrees below normal (in an ice bath), it reduces the needs of most body tissues for blood-borne oxygen. This allows the surgeon a period of eight to ten minutes to clamp off blood flow through the heart's chambers and work inside.

Heart muscle failure sometimes develops apparently because the heart muscle needs a more continuous oxygen supply than other body tissues. Even in the chill of hypothermia it soon weakens in the absence of blood and, when the surgeon restores flow through the heart, it may be flabby and fail to perform its pumping job effectively.

In their strophanthidin experiments the researchers lowered the body temperatures of a number of dogs 15 to 20 degrees below normal in ice baths. They then clamped off the blood flow into the dogs' hearts and opened their right ventricles, as if to perform surgery inside. Half of these dogs received three injections each of strophanthidin at five-minute intervals beginning fifteen minutes before circulation was interrupted. The other half did not receive the drug and served as controls.

Seventy per cent of the dogs unprotected by strophanthidin died, either during surgery or within twenty-four hours afterward, showing flabby dilated hearts and other evidences of heart muscle failure.

Less than 18 per cent of the strophanthidin-treated dogs died within twenty-four hours, and in three of these the researchers found evidence that heart muscle failure may not have been responsible.

Strophanthidin was used rather than one of the more common digitalis heart stimulants because of its comparatively fast, short-term heart tonic action. "This rapidly acting digitalis preparation given before inflow occlusion prevented the evidences of myocardial (heart muscle) failure in most of the animals and thereby increased survival," the researchers explained. "The data indicate that short term digitalization enables the myocardium to withstand temporary anoxia (oxygen deficiency)."

To date, the method has been used in hypothermia operations on more than twenty patients at the National Heart Institute.

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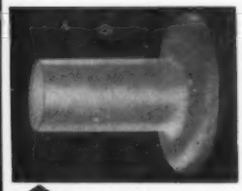

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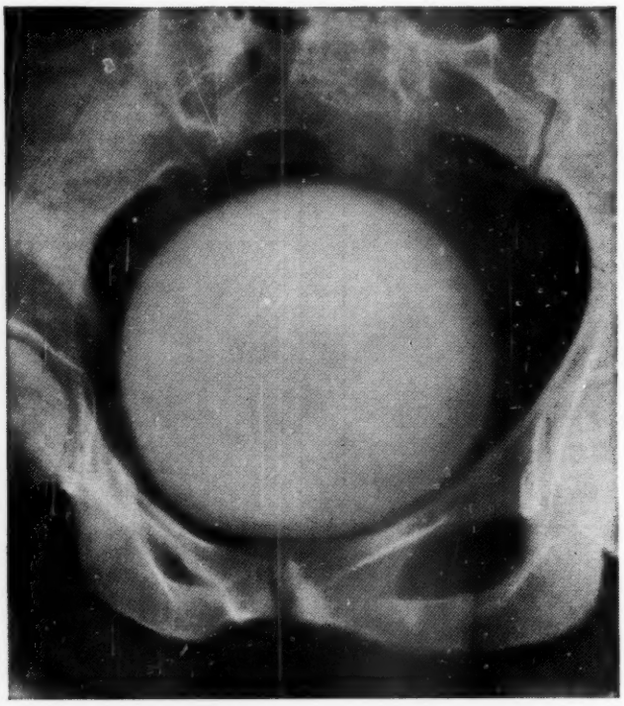

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6. Raw bony area is reinforced by slivers of cancellous bone obtained from the bone bank.

7. Incision closed.

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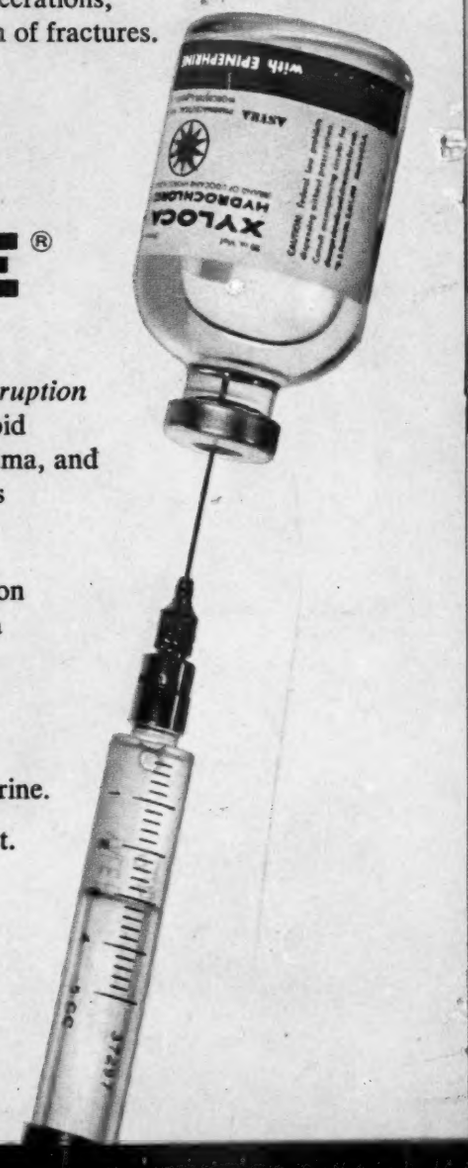

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*Southworth, J. L., and Dabbs, C. H.: Xylocaine: a superior agent for conduction anesthesia, Anesth. & Analg. *32*:159 (May-June) 1953.

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